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NAME OF SUPERVISOR/*NOM DU DIRECTEUR DE THÈSE* Dr. G. W. Fitzsimmons

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A COMPARISON OF EMG FEEDBACK AND ALTERNATIVE ANXIETY TREATMENT PROGRAMS

by

Ⓒ BRYAN A. HIEBERT

A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH
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NAME OF AUTHOR Bryan A. Hiebert

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(Signed) B.A.Hiebert

PERMANENT ADDRESS:

P.O. Box 221
Jasper, Alberta
TOE 1EO

DATED 24 November 1978

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled "A Comparison of EMG Feedback and Alternative Anxiety Treatment Programs" submitted by Bryan A. Hiebert in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Counselling Psychology.

(Supervisor)

E. E. Fox

E. W. Romaniuk

Roe C. Walley

(External Examiner)

Date 21 Nov 1978

ABSTRACT

Four cohorts of 40 subjects each were randomly assigned to one of 10 treatment conditions utilizing EMG biofeedback, cognitive monitoring training, systematic desensitization, high expectancy discussion group, or waiting list controls either in isolation or in various combinations. Subjects were obtained by announcements in the local media inviting participating in an anxiety control project. Fifty-six males and 104 females (ages 16-62, $\overline{x}$ = 29.4) completed their respective treatment programs. One-third of the sample were nonstudents.

A three-way ANOVA for repeated measures indicated that significant anxiety reductions were experienced in all noncontreal treatment conditions. Anxiety decrements for treatment groups employing EMG biofeedback were more consistent across all dependent measures (Spielberger's STAI, Cattell's IPAT Self-Analysis Form, and baseline frontal EMG). Adding desensitization or cognitive monitoring to EMG feedback did not produce a more powerful effect than using EMG feedback alone.

Demographic analysis of the data revealed significant differences in muscle tension levels between male and female subjects. Males demonstrated lower initial frontal EMG baselines, greater progress during EMG biofeedback training, and more reduction in anxiety level regardless of which treatment program was administered. Age differences were also observed in the EMG data. Baseline frontal EMG increased with age. EMG decrements during biofeedback training were greatest in the 50 years and older and 25 years and younger age categories. Subjects in the 30-39 year old range exhibited the most difficulty in acquiring control of muscle tension.

Norms are presented for 5 minute EMG baselines (N = 160).

Some implications are discussed. It would appear that EMG biofeedback focusing on awareness and control of muscle tension is more effective in producing anxiety decreases than some traditional procedures. Combining traditional anxiety treatment procedures with EMG feedback is unlikely to enhance the anxiety decrements obtained from using EMG feedback alone. Using EMG feedback to develop increased awareness of changes in muscle tension levels and cultivate a skill in willfully relaxing muscle tension appears to be sufficient to produce significant anxiety decreases without the use of adjunctive procedures. Most subjects in this study were able to apply these skills to specific environmental stressors without a specific program aimed at substituting relaxation habits for anxiety habits.

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CHAPTER I

INTRODUCTION

> So very close is the connection between the bodies
> and the minds of men, and therefore, between
> physical and mental ailments and their remedies.
>
> Alus Gellius (c. 160 A.D.)

Anxiety is a central tenet in most theories of psychopathology, and most theories agree that anxiety must be dealt with if therapy is to succeed (Reinking & Kohl, 1975, p. 595). Traditionally, anxiety has often been treated by focusing on either the cognitive aspects of anxiety, promoting insight and/or the use of cognitive coping skills as anxiety combatants, or on the behavior components of anxiety, applying the principles of learning theory to change maladaptive behavior. Proponents of biofeedback training have recently advocated treatment methods which have the potential for bridging these theoretical positions.

This study investigated the efficacy of three anxiety treatment procedures: cognitive self-monitoring, systematic desensitization, and frontal[1] EMG training. The treatment programs were conducted both in isolation and in various combinations in an attempt to understand the underlying mechanisms involved. Anxiety level was assessed using both self-report and physiological measures. Volunteers with self-diagnosed anxiety comprised the sample.

---

[1] This was referred to as frontalis training in earlier literature. However, as Basmajian (1976), Budzynski (Note 1) and others have pointed out muscle activity from other facial muscle groups (in addition to frontalis muscle activity) is registered at this site. Therefore it is appropriate to use a more encompassing label for this monitoring site.

This thesis is divided into five sections. In the first chapter a brief overview is presented. The purpose of this overview is to prepare the reader, in a general way, for the more detailed account presented later. Some basic terminology is reviewed. Some of the background thinking that led to the formulation of this research is outlined and an indication of the potential import of this study is given. In Chapter Two a more detailed discussion of relevant conceptual issues and treatment considerations is presented. In the third chapter the research design is discussed and treatment procedures are outlined. In Chapter Four the data analysis and resulting conclusions are presented. The fifth chapter contains a discussion of the implications arising from this study and some suggested directions for future research.

Overview

In this study, various combinations of three basic treatment procedures were used. These treatment programs are mentioned below along with reasons for including the programs in the present study.

Cognitive Self-monitoring

To facilitate the desensitization process, Wolpe (1969) devised the *Subjective Anxiety Scale*, an ordinal scale ranging from 0-100 in which items are separated by subjective units of disturbance (SUDS). Hiebert (1976) demonstrated that people can be taught to cognitively monitor their anxiety level using Wolpe's (1969) notion of SUDS, and that people who learn and practice this monitoring technique experience an anxiety decrement. However, these results needed to be replicated and the comparative effectiveness of SUDS monitoring and other therapeutic techniques remained to be investigated.

Systematic Desensitization

One basic assumption in systematic desensitization is that muscle relaxation is incompatible with anxiety. The goal in desensitization is to substitute relaxation responses for anxious responses in situations which usually elicit anxiety. Procedurally, a person is first taught to relax, usually using a procedure similar to that outlined by Wolpe (1958), after which they experience graduated exposure to anxiety evoking stimuli. When desensitization is complete, a subject can imagine anxiety evoking stimuli without becoming anxious. Transfer into daily life is usually sufficient to allow a person to remain relaxed in situations which formerly elicited anxiety.

Recently, the mechanism underlying systematic desensitization has been called into question (cf. Davison & Wilson, 1973). Substantial arguments have been offered which attribute the mechanism underlying desensitization to: cognitive labeling processes (Schacter, 1966; Valins, 1970), cognitive coping strategies (Lazarus, 1975; Meichenbaum, 1972, 1976), causal attribution (Weiner, 1974), extinction (Davison & Wilson, 1973), and habituation (Lader & Mathews, 1970) as well as the classical reciprocal inhibition position originally introduced by Wolpe (1958). Recently, Wolpe (1976) has stated that other factors (e.g., extinction) probably influence the desensitization process to a greater extent than he originally thought.

However, one fact remains unchallenged: systematic desensitization works. The common theme throughout substantial reviews of the literature (e.g., Davison & Wilson, 1973; Franks & Wilson, 1974; Paul, 1969; Wolpe, Brady, Serber, Agras & Liberman, 1973) is that systematic desensitization has a high probability for success when applied to the treatment of appropriate anxieties.

In the light of this demonstrated efficacy it seemed that systamtic desensitization would make a "best available alternative treatment" in a study comparing the effectiveness of anxiety treatment programs. There is an abundance of research in desensitization incorporating no contact or placebo controls (see Davison & Wilson, 1973; Paul, 1969), however there are few studies comparing desensitization to other anxiety treatment procedures of demonstrated effectiveness. Such a comparative study seemed desirable.

Biofeedback Training

"Biofeedback refers to any technique which uses instrumentation to give a person immediate and continuing signals on change in a bodily function that he is not usually conscious of" (Sullivan, 1975, p. 38).

Through biofeedback training a person learns to use the fedback biological information, first of all to recognize different bodily functions and then to gain control over them. The underlying assumption is that when a person has accurate information about a bodily function, such as muscle tension level, he can learn to control it (Karlins & Andrews, 1972; Miller, 1978). When treating anxiety, electromyographic (EMG) feedback is usually used to provide information about muscle tension levels and to teach relaxation. The goal of therapy is to increase personal awareness of muscle tension levels, and to train personal relaxation skills. When training is complete the person is able to produce his newly acquired relaxation response whenever he begins to feel anxious.

Although biofeedback programs are accredited with successfully treating anxiety (cf. Budzynski & Stoyva, 1973; Connor, 1974; Grimm, 1971; Townsend, House & Addario, 1975), the mechanisms underlying the biofeedback process are poorly understood (Miller & Dworkin, 1975).

Most of the investigations into biofeedback anxiety control procedures have been concerned with the demonstration of an effect. Once a treatment effect had been demonstrated concern was expressed over expectancy variables and the lack of systematic controlled investigations (Blanchard & Young, 1974; DiCara, 1975; Melzak, 1975; Miller & Dworkin, 1975). Recently, appeals have been made for research comparing biofeedback to established clinical procedures (Miller, 1978). It was this type of concern that prompted the present investigation.

The Problem

When one considers the theoretical foundations of biofeedback, only the most preliminary theoretical conjectures have emerged (Miller & Dworkin, 1975). Although an information theory perspective is usually adopted to explain the process underlying biofeedback training (cf. Budznski, 1973; Karlins & Andrews, 1972), it seems that other positions offer more explanatory potential.

A behavioral position. With the assistance of EMG feedback a person can learn to deeply relax, a response that is incompatible with anxiety (Karlins & Andrews, 1972; Wolpe, 1970). After training is complete, a person experiencing a situation which usually evokes anxiety can readily elicit a relaxation response instead. Repeated elicitation of the relaxation response to anxiety evoking situations will result in progressive diminuation of the tendency to respond anxiously. Through diligent repetition the habitual anxiety response pattern is gradually replaced by a more adaptive relaxation response pattern. The process is somewhat similar to that incorporated in *in vivo* desensitization. However, in a typical biofeedback program no attempt is made at hierarchy construction, or graded exposure to anxiety evolving stimuli, and no direct attempt is made to ensure that the subject does in fact remain relaxed in the presence of anxiety evoking stimuli. Wolpe (1958, 1970) claims it is necessary to maintain relaxation if anxiety is to be reduced. Therefore, most behavior therapists would likely want to combine EMG relaxation training with some other treatment technique, like systematic desensitization. If behavioral formulations contribute substantially to the biofeedback training process such a combination should demonstrate greater effectiveness than EMG feedback training alone. Since the initial work of Budzynski and Stoyva (1973) no controlled studies have emerged.

A cognitive position. Meichenbaum (1976) has offered a cognitive rationale for the biofeedback process. His basic contention is that biofeedback training ultimately occurs on a cognitive level, resulting

> in a change in the subject's perceptions, attributions, appraisals, and his internal dialogue about his ability to control his own physiological responses, cognitions, feelings and behaviors (p. 203).

Meichenbaum concludes by advocating an increased emphasis, during therapy, on the cognitive uses to which a biofeedback acquired skill can be put.

Writers who view anxiety as being cognitively precipitated (e.g., Ellis, 1973; Lazarus & Averill, 1972) could offer convincing arguments to explain the success of biofeedback in treating anxiety. From their perspective, man is by nature an evaluative being and anxiety develops when a person's cognitive appraisal mechanisms become distorted or irrational (Ellis, 1973; Lazarus & Averill, 1972). Moreover, anxiety invariably operates on a positive feedback (self-amplification) model, where a person's perception of his rising anxiety level precipitates increased anxiety. That is, as a person perceives himself becoming anxious, the very perception of the rising anxiety increases his general anxiety level. The new higher level of anxiety causes him to be more anxious, and so on (i.e., within limits, the state magnifies).

From the above cognitive perspective it could be argued that biofeedback training could provide a means for developing more accurate cognitive appraisal processes and becoming better able to recognize the physiological cues associated with an increase in anxiety level. After training the person would be better able to evaluate his anxiety level, to recognize those instances where the anxiety level is just beginning to rise but is not yet out of control, and to employ alternative coping strategies in anxiety evoking situations. The combination of awareness and new coping strategies would help to interrupt the positive feedback cycle and in fact could be instrumental in placing perceived anxiety in something similar to a negative feedback framework (Meichenbaum, 1976).

Meichenbaum (1976) claims that through biofeedback training the person learns to evaluate his cognitive reaction to his physiological responses, and that this evaluation process ultimately is carried out at a cognitive level. Therefore it would be appropriate to compare biofeedback training with other cognitive evaluative procedures like cognitive monitoring training. Moreover, if biofeedback is a primarily cognitive phenomenon, then specific training designed to promote cognitive awareness and to train cognitive evaluation procedures should enhance biofeedback training. The place of cognitive monitoring training within such a framework remains a compelling question for investigation.

The present study. The present research project was designed to address the types of questions raised above. The design incorporates many "directions for further research" advocated by DiCara (1975), Miller (1978), and Miller and Dworkin (1975). In addition to no contact controls, a high expectancy discussion condition was incorporated in an attempt to control for experimenter variables, expectancy variables, and the mystique surrounding electronic gadgetry. The design permitted efficacy comparisons between various forms of EMG biofeedback training and other anxiety treatment programs of demonstrated effectiveness. This project made possible a replication of

Hiebert's earlier work (Hiebert, 1976), enabled a comparison to be made between cognitive and machine mediated self-awareness training, and allowed for some conclusions to be made concerning the effectiveness of such training. Addressing these concerns was thought to have the potential for providing a substantial contribution in the area of anxiety treatment procedures.

CHAPTER II

THEORETICAL RATIONALE

The Construct of Anxiety

There is currently varied and discrepant opinion as to the nature and treatment of anxiety. Spielberger (1975) claims that much of the ambiguity and confusion in anxiety research seems to arise from the indiscriminant use of the word. Spielberger further suggests that progress in anxiety research will be facilitated by the adoption of terminological conventions that permit more precise communication among investigators (Spielberger, 1975, pp. 136-139). To this end, many writers (e.g., Cattell, 1972; Lazarus & Averill, 1972; Spielberger, 1975; Wolpe, 1970) have expressed concern for explicitly describing the manner in which the construct of anxiety is used. This explicitness would permit theorists to compare their substantive definitions in order to determine "whether anxiety as studied by one author has any relation to what is being studied by another" (Lazarus & Averill, 1972, p. 267).

The task of defining anxiety is not an easy one (Fischer, 1970; Lader, 1975) and ultimately no single theoretical perspective is likely sufficient to explain the wide range of anxiety reactions (Ramsay, 1975). The problem is further complicated when one addresses the issue of whether it would be more appropriate to view anxiety as an intervening variable or a hypothetical construct. It is probably safest to view anxiety as an intervening variable according to the criteria outlined by Craighead, Kazdin and Mahoney (1976). However, most writers refer to anxiety as if it were a hypothetical construct. The inherent danger in such a practice is that anxiety will begin to be thought of as an entity. As Wolpe (1970) points out, anxiety is not an entity, anxiety is a word, and it can mean whatever one ascribes. In order that the reader may understand this author's conceptualization of anxiety, a brief outline of the contributing theoretical positions is presented in this chapter. It is not the purpose of this discussion to detail different ways for conceptualizing anxiety. The interested reader is referred to the excellent accounts by Fischer (1970) and Leavitt (1967) for such a discussion. Rather, it is the intent of this author to emphasize the salient ideas that resulted in the conceptualization of anxiety underlying this study.

Behavioral Components of Anxiety

Psychologists with a behavioral orientation tend to view anxiety as a group of conditioned responses that an organism makes under certain stimulus conditions (cf. Eysenck, 1961, 1967, 1969; Lundin, 1969; Wolpe, 1958, 1973). Eysenck (1969) points out that neutral stimuli associated with pain (pain being a primary drive) give rise to a conditioned fear response very similar to the response to pain. This conditioned fear response Eysenck calls anxiety.

Kimmel (1975) distinguishes between anxiety and conditioned fear on the basis of response duration and persistance. He points out that a laboratory conditioned fear response usually lasts for only a short period of time (10-20 seconds) compared to the unpleasant emotional state of anxiety which endures for several minutes or more.

Moreover, the experience of anxiety is usually tied to some instrumental contingency designed to prevent an aversive condition. Thus, perceived increases in anxiety level are often followed by avoidance behavior. The anxiety reduction following the avoidance behavior reinforces that particular avoidance behavior and serves to emphasize the "aversive qualities" of the avoided stimulus configuration. Thus a person is seldom placed in a position to experience a nonaversive consequence to the anxiety producing situation. This results in an anxiety response being very resistant to extinction, whereas a conditioned fear response rapidly extinguishes upon removal of the unconditioned stimulus.

Wolpe (1958, 1969, 1973) agrees that anxiety is often conditioned to environmental stimuli, but he emphasizes the importance of the learned behavioral characteristics of an anxiety response rather than the drive reduction characteristics *per se*. According to Wolpe (1958), as drive states arise, they excite overt action. If the resulting behavior is adaptive, the drive state is dissipated. If the resulting behavior is unadaptive, the excitement is sustained, and becomes labeled anxiety. This state of sustained excitement often becomes conditioned to environmental events through temporal contiguous association with these events. The result is an increased tendency for persistent maladaptive behavior which is characteristic of most neuroses. Wolpe (1973) further holds that anxiety is largely proprioceptively precitipated; i.e., anxiety is precipitated by a perceived increase in muscle tension, which is invariably the result of a person's prior conditioning.

The above discussion illustrates the ease with which semantic differences could give rise to apparent theoretical discrepancies. However, several commonalities exist. From a behavioral perspective anxiety is generally agreed to be accompanied by an increase in physiological arousal, which becomes conditioned to environmental stimuli. There is usually a strong, maladaptive behavioral component in an anxiety response, and there is generally considered to be some qualitative difference between fear and anxiety, although the characteristics of this difference vary between theoreticians.

Cognitive Components of Anxiety

Most cognitively oriented psychologists acknowledge the behavioral aspects of anxiety, but are quick to point to the existence of an organism which intervenes between the environmental stimulus and the behavioral response. Some writers (e.g., Schacter, 1966; Valins, 1966, 1970) suggest that behavior is based on the perception, not just the occurrence, of environmental situations. Others (Ellis, 1962, 1973; Lazarus, 1974, 1975; Lazarus & Averill, 1972; Meichenbaum, 1972) view cognitive processes as playing a more direct role in precipitating emotional experience. Recently, writers like Lazarus (1974, 1975) and Meichenbaum (1972, 1975) have begun to emphasize the role of cognitive coping mechanisms in anxiety treatment.

According to Schacter (1966) and Valins (1966, 1970) perceived physiological changes give rise to cues which are cognitively processed as feelings. These feelings in turn result in further cognitive activity geared towards attempting to identify the eliciting situation. When an individual experiences a state of physiological arousal for which he has no immediate explanation, he will label the state and describe his feelings in terms of the cognitions available to him (Schacter, 1966), i.e., the same perceived state of physiological arousal could be labeled as joy, anger, fear, or whatever, depending on the individual's perception of the environmental situation. If a state of physiological arousal is experienced and no explanation as to the precipitating stimulus is immediately available, the individual is likely to attach uncommon or inappropriate labels to environmental stimuli in an attempt to provide a higher degree of understanding for his aroused condition. This is the manner in which bizarre or unadaptive behaviors, which are a part of most anxiety reactions, develop.

The framework discussed above bears a substantial resemblance to the position advanced by Ellis (1962, 1973). His school of thought holds that a person's emotional or behavioral reaction in a situation is primarily the result of the person's cognitive appraisal of the situation, rather than the specific situational stimuli *per se*. When a person's perceptions/beliefs/appraisal of the situation become inaccurate or irrational or somehow distorted then maladaptive behavior (or emotion) results. The emotional experience resulting from these misconstrued perceptions is invariably self-amplifying (i.e., a positive feedback loop is established) and self-defeating (Ellis, 1973, p. 178). From his perspective, the accurate perception and appraisal of arousal inducing events becomes crucial.

Lazarus and Averill (1972) elaborate the same point. They state that man is by nature an evaluative being, searching his environment for what is needed or desired, evaluating each input with respect to its relevance and significance (p. 242). This cognitively based appraisal process mediates the organism's reaction to environmental stimulation, with the emotional reaction, and the resulting behavior, being a result of the cognitive appraisal rather than the environmental stimulation *per se*. Anxiety is largely the result of appraisal errors with respect to the consequences of future events, the degree of ideational threat or the degree of ambiguity in a perceived source of threat. Lazarus (1974) points out that much of the experience of anxiety is anticipatory, i.e., the emotional experience occurs prior to the occurrence of an event. A person perceives a situation, anticipates the consequences, evaluates the degree of threat, then enacts some behavioral strategy usually designed to reduce the level of anxiety.

Meichenbaum (1972, 1975) offers additional support to the above position. He envisions two main constituents of anxiety: emotionality, characterized by heightened arousal, and worry, characterized by self-degenerating thoughts and undue concern over performance. Because of the attention it demands, worry interferes with performance, even though heightened arousal could be facilitative. A perceived arousal state that becomes labeled anxiety is usually followed by feelings of personal inadequacy and denigrating self-verbalizations which increase the anxiety level (Meichenbaum, 1976). In this way the very perception of a rising level of anxiety produces more anxiety, and a positive feedback loop is instituted. Meichenbaum (1972) claims that treatment techniques like systematic desensitization are aimed at the arousal dimension of anxiety, and that coverant (covert operant) conditioning procedures are necessary to reduce the worry dimension. (Simkins, 1971, refers to coverants as "the operants of the mind," which Meichenbaum claims follow the same principles as overt behavior modification.)

Lazarus (1974) offers a potential framework for combining the above perspectives. He postulates three components of emotional experience (including anxiety): A physiological component, characterized by changes in arousal level; a subjective component, involving the perception, labeling, and evaluation of internal and external environmental stimulation; and a behavior component, having instrumental as well as expressive characteristics. Whether or not a particular emotional experience would become labeled anxiety would depend on the interaction between these three components.

All of the orientations mentioned above could be subsumed with Lazarus' (1974) framework. All the positions outlined above view anxiety as being accompanied by a perceived state of heightened arousal. Anxiety occurs when this perception becomes somehow distorted, or associated with feelings of personal inadequacy, and triggers off maladaptive or inappropriate behavior. Thus anxiety is seen as an interfering emotion. As a result, anxious people tend to perceive an exaggerated degree of threat in many environmental situations, and an overabundance of situations precipitating this perception (Ramsay, 1975). The incorrect or inappropriate perception of the situation is a prerequisite to the experience of anxiety, and some procedure for realigning perceptions is considered to be a central part of any therapeutic intervention aimed at reducing anxiety.

One cannot overlook the prominent position occupied by the individual in the above discussion. Although he admits that the empirical basis for this claim is still somewhat uncertain, Lazarus (1975) holds that a widespread movement is beginning towards the position that cognitive processes intervene between a person's adaptive transactions with the environment and his emotional reaction to it.

Physiological Components of Anxiety

One underlying theme in the above discussion is that anxiety is closely associated with physiological arousal. (The author recognizes the controversial and wide ranging nature of the arousal concept. In this paper arousal is used in a general manner to describe increases in certain types of physiological activity, i.e., increased activity in the sympathetic nervous system and the skeletal musculature.) Some go so far as to claim that arousal and anxiety are the same (Wolpe, 1958) or, modified somewhat, that when only physiological variables are considered, there is little or no difference between anxiety and arousal (Gray, 1971; Leavitt, 1967; Wolpe, 1958).

Budzynski (1973, Note 1) and Raskin, Johnson and Rondestvedt (1973) point out that anxiety is accompanied by increases in physiological arousal. Although individual arousal patterns may differ, the changes are usually in the direction of increased muscle tension levels, skin conductance, blood pressure, heart rate, respiration rate, pupilary dilation, and peripheral vasoconstriction (Budzynski, 1973; Germana, 1974). The particular physiological response may vary from person to person, but the ideosyncratic response tends to remain fairly constant across anxiety inducing stimuli. Thus a person who responds to an anxiety evoking stimulus with increased muscle tension will tend to react with muscle tension increases regardless of the specific anxiety stimulus. Budzynski (1973) points out that these anxiety reactions are more or less autonomous in that they occur automatically and are often below the individual's level of awareness (p. 86). Furthermore, anxious individuals tend to react to anxiety evoking stimuli with exaggerated increases in arousal, which are sustained for prolonged periods, and which dissipate slowly as compared to the general population (Budzynski, 1973, Note 1; Budzynski, Stoyva, Adler & Mulloney, 1973; Malmo, 1970).

As noted above, the increases in arousal may be manifested in numerous ways. Increased muscle tension is one of the more commonly recognized components of an anxiety reaction. Malmo (1975) states that overactivation of the skeletal musculature is clearly involved in chronic anxiety (p. 12). Buck (1976) concludes that muscle tension in irrelevant muscles (those not engaged in the task at hand) seems to increase with anxiety. Abnormally prolonged contraction of particular muscle groups is common in patients with high anxiety (p. 432). Smith (1973) found that frontal EMG correlated significantly with anxiety level, as measured by the IPAT Self-Analysis Form (Cattell, 1957). Bearing this in mind, anxiety treatment usually involves the cultivation of a low arousal response that can be used to inhibit anxiety (Budzynski, 1973, Note 1, Raskin *et al.*, 1973; Stoyva & Budzynski, 1974). The above discussion of the role of muscle tension in an anxiety response supports the common use of muscle relaxation as a means for training a low arousal response.

Some writers (e.g., Malmo, 1970; Whatmore & Kohli, 1974) postulate that the exaggerated reaction referred to above, is the result of impaired physiological control mechanisms. Malmo (1970) claims that the increased muscle tension levels in anxious individuals are the result of a neurological impairment in the mechanisms that control muscle tension. Whatmore and Kohli (1974) posit that all functional disorders (anxiety reactions included) are the result of dysponesis: a physiopathologic state caused by errors in action potential output. Anxiety is identified as dyslimbic fear, where bracing errors and representation errors result in excessive activation of fear limbication, usually resulting in inappropriate behavior, and often becoming conditioned to environmental stimuli. For these authors, treatment consists of specialized training in order to identify and correct the signalling errors contributing to dysponesis.

Regardless of theoretical orientation, the physiological components of anxiety occupy an important position. It is now "widely accepted that . . . anxiety reactions are characterized by feelings of apprehension, tension, and activation of the autonomic nervous system" (Spielberger, 1975, p. 129). Furthermore, it seems that some means of controlling or inhibiting these physiological reactions is an important part of an anxiety treatment program.

Synthesis

The integration of differing theoretical conceptualizations of anxiety is difficult and seldom attempted (Fischer, 1970). However, such integration is necessary if one wishes to achieve a conceptual basis that permits cross theoretical comparison (Spielberger, 1975). The foregoing theoretical positions have all influenced the notion of anxiety underlying this investigation. However, the contribution has been differential because of the incompatible nature of several aspects of each position.

Spanos and Barber (1974) object to the usual tendency in cross theoretical comparisons to emphasize differences in theoretical orientation, saying this tendency fosters a distorted view of the research area, obscures areas of agreement, and often leads to polemical battles of little practical value. In view of this objection an attempt was made to tease out conceptual commonalities and to weave them into a compatible conceptual framework from which to view anxiety.

Probably the most obvious common point is that virtually all theoretical positions allow that anxiety is accompanied by increases in physiological arousal, (although most theorists would be quick to add that anxiety is more than just a state of physiological arousal). The increase in arousal is invariably followed by behavior designed to reduce the arousal level. This behavior is often a maladaptive or inappropriate overt avoidance response pattern, however, most theoretical positions could accommodate the existence of maladaptive covert response patterns. These covert patterns include cognitive appraisal mechanisms, cognitive labeling mechanisms, negative expectations, negative self-denigrating subvocalizations, which function within a positive feedback framework. The emotional experience of anxiety is generally held to be unpleasant, and reducing the intensity of an anxiety reaction is generally considered to be rewarding. Most theorists agree that anxiety often becomes associated with specific environmental events. Anxiety generally appears to have some subjective idiosyncratic component because of the variation in response patterns across subjects. Anxiety generally follows the exaggerated perception of threat in an environmental situation. Anxiety can therefore be distinguished from fear on the basis of the degree of objective threat a situation affords.

Definition. The notion of anxiety underlying this study was formed from the above considerations. Anxiety is viewed as an unpleasant, interfering emotional state recognized by increases in physiological arousal. The experience of anxiety is usually characterized by maladaptive behavior (overt avoidance behavior or covert self-denigration) which tends to be self-sustaining or even self-amplifying. Anxiety producing situations are typically perceived as containing an exaggerated degree of threat: things seem worse than they really are. This experience of anxiety usually becomes associated with certain environmental stimuli in such a way that certain things in a person's life will predictably, repeatedly, precipitate anxiety.

The Treatment of Anxiety

Just as there are a variety of theoretical conceptualizations of anxiety, so too there is great variation in the treatment approaches that are used to combat anxiety. Lazarus (1974, 1975) describes anxiety treatment programs as fitting into two categories: those that involve altering a person's relationship with his environment and those that alter a person's reaction to his environment. The former approaches deal directly with the problem generating the anxiety, while the latter approaches are concerned with reducing the intensity of the visceral or motor disturbances that occur when the problem is encountered. It might be appropriate to label the first category "alteration approaches," for the basic goal is to replace the person's habitually anxious response patterns with more adaptive response patterns, and to label the last category "control approaches," for the basic goal is to diminish (or control) subjective response to stimuli which elicit an anxiety reaction.

The present investigation utilizes both types of treatment programs. This discussion will focus on describing the various treatment procedures in general terms and evaluating some of the related research. Subsequent sections of this thesis will elaborate the research procedures in more detail.

Biofeedback

Feedback systems operate on their ability to detect changes in the environment of their operation, and to make the necessary internal adjustments so that functions remain both optimal and continuously appropriate to the demands of their environment (Brown, 1974, p. 4). "The biofeedback idea is a simple one: knowledge of the dynamic status of biological systems will enable voluntary control over such a system" (Paskewitz, 1975, p. 371). Typically some physiological variable is sensed through electrodes, amplified, filtered, processed, and displayed to the subject as a series of clicks, a variable tone, a print on a graph or a set of lights that vary according to the status of the function being monitored. The information thus represented allows the subject to enter a feedback loop with his internal environment where the equipment forms a significant information pathway within the total system. Through biofeedback a person learns to use the fedback biological information first of all to recognize different bodily functions, and then to gain control over them (Brown, 1974; Budzynski, 1973; Karlins & Andrews, 1972).

Biofeedback training involves three related (nondistinct) stages (Budzynski, 1973; Karlins & Andrews, 1972; Meichenbaum, 1976). In the first stage, the primary focus is on awareness: the person learns to recognize the physiological parameter being monitored. By using the information displayed by the monitoring equipment the subject develops his own idiosyncratic indicators of a particular bodily function such as heart rate, skin temperature, brainwave pattern (EEG), muscle tension level (EMG), or galvanic skin response (GSR). Specifically, the person learns to become adept at detecting changes in the physiological function being monitored.

In the second stage the person learns to control the bodily function being monitored. He is told to not think about his heart rate or his muscle tension but instead to direct his attention at the feedback monitor and try to change the tone, slow down the clicks or make the light come on. When people direct their attention at changing a subtle behavior, like heart rate or muscle tension, they frequently achieve the opposite of the desired result. However, by directing attention at the feedback monitor and attempting to change the rate of clicking or the pitch of the tone, most people can learn to control the bodily function being monitored (Karlins & Andrews, 1972).

The strategy underlying the control phase is variously referred to as "passive volition" (Sargent, Walters & Green, 1973), "effortless trying" (Green, Green & Walters, 1969), or "passive concentration" (Luthe, 1972), for when an individual stops trying and just lets it happen he can produce muscle relaxation, or alpha brain waves at will. The length of time required varies considerably, with subjects requiring as few as two sessions (Gallon & Padnes, 1976) or as many as forty sessions (Raskin *et al.*, 1973) to learn control. Sargent *et al.* (1973) report that younger subjects train more quickly, and May and Weber (1976) report that subjects demonstrating the most severe symptoms learn more effective control. Controlled research on these matters remains to be undertaken. Some researchers (Herzfeld & Taub, 1976; Sheridan, Boehm, Ward & Justesen, 1976) report the facilitative effect of autogenic phrases, and other suggestion procedures (e.g., slide presentations, relaxation tapes) but others (Coursey, Frankel & Gaarder, 1976; Reinking & Kohl, 1975; Wilkinson, 1976) report that the use of these techniques has little facilitative value. At present the necessity of these "logical training facilitators" is at best controversial.

During the third stage of training the subject learns to transfer his control from the laboratory into his daily life. The process is known as weaning (Weiss, 1975) and results in the person being able to willfully produce the required response without the aid of the feedback monitor. This is usually accomplished by gradually reducing the volume of the feedback signal, having several "no feedback" periods during a training session, having the person relax under baseline (no external feedback) conditions, or having the person institute periods of wilful relaxation into his daily routine. These procedures ensure that a person is not reliant on the feedback monitor to produce a physiological response, rather that the person has developed his own internal feedback cues on which to gauge the successfulness of his attempts at volitional physiological self-regulation.

Typically, a biofeedback anxiety program uses the feedback loop to cultivate a low arousal condition (cf. Stoyva & Budzynski, 1975) which can later be used as an anxiety inhibitor. Some therapists use alpha feedback (cf. Benjamins, 1976), or respiratory feedback (cf. Grimm, 1971), however electromyographic (EMG) feedback is more commonly used (cf. Budzynski & Stoyva, 1973; Leaf & Gaarder, 1971). Although much of the research in anxiety incorporates GSR as a physiological indicator of anxiety (e.g., Hyman & Gale, 1973; Kimmel, 1975; Lader & Mathews, 1970) GSR feedback is not widely used in the biofeedback treatment of anxiety, mainly because of replication problems and inconsistent results (Blanchard & Young, 1974).

The decision to use EMG feedback in anxiety treatment is appealing on logical grounds for muscle relaxation has long been considered to be incompatible with anxiety (Grossberg & Wilson, 1968; Karlins & Andrews, 1972; Luthe, 1972; Wolpe, 1958, 1973). Reinking and Kohl (1975) report that muscle activity is the best single physiological correlate of anxiety. Usually the forehead area is used as a training site because frontal EMG is believed to be an indicator of general relaxation (Leaf & Gaarder, 1971).

There is some controversy over the use of frontal EMG as a good indicator of general body relaxation (Alexander, 1975; Wilkinson, 1977). Alexander (1995) found no evidence that frontal EMG training generalized to untrained monitoring site in the forearm or lower leg. However Alexander's extensive subject screening system, his brief and frequently interrupted training session, and his method of integrating the EMG signal (sequentially switching between the three monitoring sites rather than monitoring simultaneously), may have biased his findings.

In spite of objections to the contrary, there is substantial support for the use of EMG for relaxation training (Budzynski, 1973; Budzynski & Stoyva, 1969; Budzynski, Stoyva & Adler, 1970; Leaf & Gaarder, 1971; Reinking & Kohl, 1975; Stoyva & Budzynski, 1975) as well as in conjunction with systematic desensitization (Budzynski & Stoyva, 1973). Moreover, many reports support the claim that frontal EMG training is more efficacious in lowering muscle tension than traditional procedures for producing general body relaxation (cf. Hayns, Moseley, & McGowan, 1975).

Although there is beginning to be a reasonable research base establishing the effectiveness of EMG assisted relaxation training, there have yet to be any published reports of controlled research into the comparative efficacy of an EMG anxiety treatment program and systematic desensitization; either comparing the two procedures in isolation or in combination (Budzynski, Note 2). Blanchard and Young (1974) report that the combination of EMG biofeedback training and home relaxation practice is effective in reducing muscle tension. This conclusion has obvious implications in the area of anxiety treatment. There are now several research reports comparing EMG biofeedback training with other relaxation training procedures (cf. Coursey, *et al*., 1976; Friedman & Papsdorf, 1976; Reinking & Kohl, 1975; Coursey & Chao, Note 3) but a careful search of related literature failed to uncover any such comparative studies conducted within an anxiety treatment context. Currently, there are a substantial number of case study reports attesting to the effectiveness of biofeedback training in a variety of treatment settings. Some writers (e.g., Miller, 1978; Miller & Dworkin, 1974) see such case study reports as a necessary first step in demonstrating treatment efficacy. However, Miller (1978) advocates two further steps in the evaluation of clinical treatment procedures: (a) controlled comparisons with the best available alternative techniques, and (b) broad clinical trials on large samples under the conditions to be expected in general use. Clearly, there is need in the area of anxiety treatment, for carefully controlled comparison studies conducted under conditions that closely approximate a clinical setting.

Cognitive Self-monitoring

Hiebert (1976) developed a program for training subjects in the cognitive self-monitoring of anxiety level. The program consisted of teaching subjects to use Wolpe's (1969) notion of SUDS to quantify anxiety in their ongoing day-to-day environment. During two treatment sessions subjects received instruction and examples of the SUDS monitoring concepts. Subjects also practice the use of SUDS monitoring via a slide presentation depicting scenes that would typically elicit a broad range of anxiety reactions. As part of a home practice assignment, subjects were instructed to monitor their SUDS levels first at hourly intervals, then continuously throughout the day. Subjects learning this monitoring procedure reported an anxiety decrement that was maintained over a two week follow-up period. Since then the author has used the idea of cognitive monitoring clinically, with more than 300 persons. Clients consistently report benefits from this type of systematic self-awareness training: increased feelings of self-autonomy, progressively lower anxiety levels, greater awareness of low anxious or non-anxious situations, and a greater tendency for things that "bothered them" to "no longer have any effect." Although the results have not been replicated in a controlled setting, the initial investigation holds promising treatment implications.

The basic assumption underlying the above discussion is that learning to cognitively self-monitor anxiety level results in a person learning to recognize his own idiosyncratic indicators of an anxiety reaction which in turn provides therapeutic benefit. The position is similar to the first stage of biofeedback training (namely learning to recognize the subjective indices of the parameter being monitored) except that it occurs solely on a cognitive, non-machine-mediated level. From Hiebert's (1976) initial investigation it would appear possible to parallel the first stage of biofeedback on a strictly cognitive level. However, comparison between machine and cognitive mediated self-awareness training would be necessary before such an inference could be drawn. The present study made such a comparison possible.

Most biofeedback therapists include some treatment phase where clients are instructed to continuously monitor their own tension levels, a process which Meichenbaum (1976) claims must ultimately be carried out on a cognitive level. Even Whatmore and Kohli (1974) advocate that orthoponesis (the process of teaching patients to identify and correct signaling errors) not only include laboratory training in the detecting and correcting of signaling errors, but also training in the routine monitoring of personal reaction to the environment. (Unfortunately they do not indicate how this latter procedure is developed.)

According to the information theory perspective maintained by most biofeedback therapists, precise information about anxiety level (or some physiological correlate of an anxiety reaction) facilitates learning to control one's anxiety responses. If this increase in self-awareness actually facilitates an anxiety decrement, then a cognitive monitoring program could also facilitate a decrease in anxiety level. Relatively little research has been conducted to test such a proposition. Furthermore, the comparative effectiveness of machine-based and cognition-based self-monitoring procedures remained to be investigated. The present study was formulated, in part, to fulfill these needs. It represents an attempt to replicate earlier findings, and explore the parameters of the cognitive self-monitoring process. Exploring the possibility of paralleling biofeedback training on a purely cognitive (non-machine-mediated) level had potential theoretical and practical benefit. Furthermore, comparing the results of machine-mediated and cognitive monitoring procedures could provide useful clinical information.

Systematic Desensitization

Systematic desensitization (Wolpe, 1958) is perhaps the most widely researched of the behavior therapy procedures (cf. Davison & Wilson, 1973; Franks & Wilson, 1974). Procedurally the subject first learns to relax, a response generally considered to be incompatible with anxiety. While relaxation training is proceeding, a fear hierarchy (an ordered list of anxiety evoking situations associated with a particular phobia or anxiety reaction) is prepared. Next the subject imagines himself in each situation described in the fear hierarchy, beginning with the least anxiety evoking and proceeding systematically through the list while remaining relaxed. The criteria for progressing from one hierarchy item to the next is the ability to remain completely relaxed while visualizing a given scene. When a subject can imagine himself in a given situation and experience close to zero SUDS of anxiety he proceeds to the next item of the hierarchy. When desensitization is complete, the subject can imagine himself in the situation that originally generated the most anxiety and remain completely relaxed. Transfer into daily life is usually sufficient to enable the person to experience the situations in vivo without becoming anxious.

According to Wolpe (1958) relaxation and anxiety are reciprocally inhibiting. As the strength of the situation-relaxation habit is increased (through the repeated imagining of a hierarchy scene while remaining relaxed) the situation-anxiety habit is progressively diminished in strength. This is a classical counter-conditioning paradigm where a relaxation response is ultimately substituted for an anxiety response. As long as the anxiety level remains close to 0 SUDS during the visualization of the hierarchy scenes, anxiety is effectively inhibited (Wolpe *et al.*, 1973).

Wolpe's (1958) original explanation of the mechanism underlying systematic desensitization still receives wide-spread acceptance. However the theoretical underpinnings of systematic desensitization have recently been called into question (cf. Davison & Wilson, 1973; Franks & Wilson, 1974). The necessity of relaxation has been refuted (Walters, McDonall & Koresko, 1972), the role of expectancy has been challenged (Wilkins, 1971), the facilitative effect of hierarchy construction has been examined (Emery & Krumboltz, 1967), the role of visualization has been questioned (Wilkins, 1971), and the facilitative effect of repeated scene presentation has been emphasized (Davison & Wilson, 1973). Group desensitization procedures (cf. Richardson & Suinn, 1973) have become popular and automated self-desensitization procedures (cf. Gershman & Clauser, 1974) have begun to be used. Both of these variations hold theoretical implications for Wolpe's procedure. It seems at least safe to say that the mechanisms underlying desensitization may be different from those Wolpe (1958) originally postulated (Davison & Wilson, 1973).

One consistent observation from the majority of the research is that systematic desensitization does work; subjects who receive this treatment usually experience an anxiety decrement. Consequently systematic desensitization seems like a good "best-available-alternative-treatment" to use in a comparison study.

Connor (1974) expressed concern that the methodological implications arising from the aforementioned research have not been widely incorporated into clinical desensitization procedures. Furthermore, Wolpe *et al.*, (1973) emphasize that the therapist must constantly monitor the felt and observed muscle tension in their patients during the desensitization process. The implied role of some device to assess muscle tension changes is clear in Wolpe's statement. EMG feedback could be used in such a context as an objective indicator for determining progress through a hierarchy, or as a signalling device to inform the therapist or patient of muscle tension increases. However, since the initial work of Budzynski and Stoyva (1973) no controlled studies have emerged.

In a research setting, physiological monitoring of the subject during systematic desensitization is relatively common. Most researchers use GSR (cf. Mathews, 1971; Lang, Melamed & Hart, 1970), based on the argument that GSR and heart rate are indicators of autonomic arousal, and anxiety is an autonomic reaction. The reader is reminded of Blanchard and Young's (1974) observation that GSR biofeedback research has been fraught with inconsistency and replication difficulties. Moreover, if one accepts the relationship between anxiety and muscle tension postulated by Wolpe (1958, 1970) then EMG would be a more logical parameter to monitor. Grossberg and Wilson

(1968) and Lang *et al.* (1970) included EMG in their parameters being monitored and found that EMG was less reactive, and therefore less suitable for consideration. However, it should be noted that Lang *et al.* (1970) used forearm EMG which is a less suitable monitoring site, and Grossberg and Wilson (1968) failed to indicate their EMG monitoring site. Furthermore, the latter authors suggest that different procedures may have to be developed for scoring EMG data because it is not an autonomic reaction. Other researchers (Reinking & Kohl, 1975) have suggested that EMG is the best single correlate of anxiety and its use as an anxiety indicator is becoming more widespread (Budzynski, Note 1).

Certainly the above discussion points to the need for more research in systematic desensitization. Combining frontal EMG with desensitization could provide support for the reciprocal inhibition hypothesis posited by Wolpe (1958). Monitoring EMG during desensitization would provide data on changes in muscle tension levels during desensitization. Combining desensitization with formal SUDS training could lend support for cognitive desensitization position (similar to Meichenbaum, 1972) especially if EMG levels did not change during desensitization. Attribution based explanations for desensitization (cf. Valins, 1966; Weiner, 1974) would imply that merely monitoring anxiety level might be sufficient to reduce anxiety, i.e., the increased self-awareness accompanying SUDS training might result in subjects developing their own specific coping mechanisms. The comparison between such monitoring and traditional desensitization merits investigation.

The Role of Expectancy

Concern has been expressed over the role of expectancy variables in biofeedback training (DiCara, 1975; Miller, 1978) and systematic desensitization (Kazdin & Wilcoxon, 1976). Appeals have been made to include adequate expectancy controls in biofeedback research. This is difficult as Miller (1978) points out: "Because most biofeedback procedures are so impressive, a less impressive procedure is not an adequate control" (p. 387). For this reason attention placebo groups may not constitute an adequate expectancy control: they do not engender as much anticipation of positive therapeutic outcome as procedures like EMG biofeedback, or systematic desensitization.

There is a popular notion that emotional experiences like anxiety stem from some imbalance in underlying personal dynamics. If one adheres to this psychodynamic proposition then anxiety can best be treated by attacking the roots of the problem rather than merely focusing on the overt anxiety symptoms. Although the above notion is not without theoretical support (cf. Holland, 1973; Meador & Rogers, 1973; Mosak & Dreikers, 1973; Perls, 1969), there seems to be a dearth of systematic research to support the existence of such underlying dynamics. It seemed therefore that a satisfactory high expectancy condition could be attained by adopting a discussion-group format which addressed these underlying concerns. The program for such a group could be accomplished by consulting with psychologists having existential, transactional analysis, gestalt or client centered orientations, to find out the types of concerns and issues they would address in an anxiety treatment group. A list of such concerns might include: self-perception, beliefs, values, goals, self-autonomy, other-directedness, self-destiny, and self-responsibility. These issues could then form a "hidden agenda" for a discussion group focusing on altering underlying factors that trigger off an anxiety reaction so as to produce lasting relief from anxiety. In this way, a high expectancy condition could be attained which would approximate the potency of a biofeedback program.

Research Implications

As has been previously described, there is current controversy surrounding the question of why persons taking part in a biofeedback training program usually experience therapeutic effect. Research incorporating the considerations mentioned above could have many explanatory implications. If counter conditioning is the mechanism underlying biofeedback treatment of anxiety, then combining biofeedback training with systematic densensitization should produce a more positive effect than using either procedure in isolation. Alternatively, if the combination does not produce a greater anxiety decrement than biofeedback alone, then a stronger case could be made for an explanatory position based on information theory.

If the awareness phase of biofeedback training is an important component, then exploring and comparing alternative self-awareness procedures, like cognitive monitoring training, becomes viable. Moreover, since monitoring one's arousal phenomenon, providing a cognitive framework for biofeedback training should have a facilitative effect. Specifically, combining the SUDS notion with biofeedback training should result in a greater anxiety decrement than either used in isolation.

In the light of the directions for future research mentioned earlier (DiCara, 1975; Miller, 1978) a study investigating the effectiveness of EMG biofeedback, cognitive self-monitoring and systematic desensitization would be of clinical import. A design incorporating high expectancy and control conditions would strengthen the clinical inferences that could be drawn. Monitoring muscle tension levels during relaxation training and desensitization could provide information concerning the accuracy of self-reported anxiety experiences and the role of expectancy in anxiety treatment. Using a variety of dependent measures would enable some statement to be made regarding the most appropriate measures to be used in research of this nature. These concerns had a major impact in the formulation of the present study.

Hypotheses

The above mentioned considerations can be more explicitly stated as testable hypotheses.

1. Persons receiving treatment in the form of EMG biofeedback training or instruction in cognitive monitoring will experience an anxiety decrement, as evidenced by: (a) pretreatment-post-treatment score comparisons on all anxiety measures, (b) treatment-control (placebo) post-treatment score comparisons, and (c) treatment-control (no contact) post-treatment score comparisons.

2. The anxiety reduction hypothesized in #1 will be retained

over time, as evidenced by the self-report of a random sampling of subjects one month after the termination of treatment.

3. Biofeedback training and instruction in cognitive monitoring will be equally effective in producing an anxiety decrement, as evidenced by inter-group comparison of post-treatment anxiety level.

4. People who receive biofeedback training within the cognitive framework provided by cognitive monitoring training will experience a greater anxiety reduction than people who receive either biofeedback training or instruction in cognitive monitoring in isolation, as evidenced by inter-group comparison of post-treatment anxiety level.

5. Persons who receive systematic desensitization as an adjunct to biofeedback training or cognitive monitoring training will experience a greater anxiety reduction than persons who receive biofeedback training in isolation, as evidenced by inter-group comparison of post-treatment anxiety level.

6. People who receive systematic desensitization as an adjunct to biofeedback training will experience a greater anxiety reduction than persons who receive traditional systematic desensitization, as evidenced by inter-group comparison of post-treatment anxiety level.

The research design, sampling, assessment, treatment and analysis procedures by which the above hypotheses were tested, will each be discussed in subsequent chapters of this thesis.

CHAPTER III

PROCEDURE AND DESIGN

The hypotheses listed in the previous chapter could have been tested in a variety of ways. In this chapter, the procedural characteristics of the present study are discussed. Initially, the research design is elaborated. The sampling procedures, sample constitution, and physical facilities are discussed next. The dependent measures used in this study are then briefly discussed, and finally the clinical procedures for each treatment group are detailed.

Research Design

The research design used in this study was essentially a two stage, five group, repeated measures design (see Figure 1).

During stage one the primary emphasis was on awareness of anxiety. In stage two, subjects either continued with the treatment format commenced in stage one or entered a habit substitution condition incorporating systematic desensitization. All treatment groups (groups 1-4) received six 50 minute treatment sessions. All subjects were pretested at the beginning of session one (see T_1 in Figure 1). All dependent measures were also administered at the beginning of session three (commencing stage two, see T_2 in Figure 1), and again as a posttest during session six (see T_3 in Figure 1). Waiting list control subjects (group 5) were told that it was necessary to obtain

T_1 T_2 T_3

Group Number (Factor A)		Treatment Procedure (Group Number)
1	EMG Biofeedback Training	Continue training (11)
		Systematic desensitization (12)
2	Cognitive Monitoring	Continue monitoring (21)
		Systematic desensitization (22)
3	EMG Biofeedback Training and Cognitive Monitoring	Continue monitoring (31)
		Systematic desensitization (32)
4	High Expectancy Discussion Group	Continue placebo interaction (41)
		Systematic desensitization (42)
5	Waiting List Control	Continue no contact (51) (52)

Figure 1. Schedule for testing and treatment. Anxiety measures were administered at T_1, T_2, and T_3.

Note: The group number in parentheses is used to identify treatment procedures in subsequent pages of this thesis.

a stable indication of their anxiety level before treatment could begin. These subjects completed all dependent measures on the same days as subjects assigned to treatment conditions. After the three assessments referred to above, waiting list control subjects were assigned to biofeedback practicum students for treatment. All treatment programs were conducted in groups of four. Therefore at any one time there were potentially 40 subjects in the study. When one group of 40 subjects (a cohort) finished their respective treatment programs there would be a one week break, after which another group of 40 subjects (cohort) would commence treatment. Thus each cohort was a replication of an earlier group. In all, five such replications were conducted creating a potential sample of 200 subjects. However, some of the cohorts were only partially filled and in the end 173 subjects volunteered to take part in the project. A random sampling of subjects from each cohort were contacted about a month after the termination of treatment for follow-up purposes.

Sampling Considerations

Subjects were obtained from announcements in the local media and to large undergraduate classes inviting participation in an anxiety control project. A press release was issued through the University public relations department to all media north of Red Deer. The press release stated that there was a project comparing different anxiety control procedures being conducted at the University of Alberta and that people were invited to telephone the number listed if they wished to participate. The class announcements consisted of an abbreviated form of the press release. Any questions concerning details of the various programs were answered by saying that such details would bias the research and therefore would not be discussed until the project was completed. The classes were chosen to represent a wide sampling of students from all departments and faculties.

It was initially planned to conduct four replications of the research design before Christmas. However, the third cohort was only partially subscribed and no one volunteered for the fourth time period. Therefore, it was decided to issue another press release in early January and conduct two more replications of the study in order to obtain sufficient numbers to make data analysis feasible. Of the 173 subjects commencing the project, 162 completed their respective treatment programs. This represented a 6.36% drop-out rate. Basically, all subjects who withdrew from the program were feeling too anxious to continue treatment. The reasons for non-attendance ranged from pressure over term papers and exams, to marital problems, to apprehension about student teaching, to hassles at work. In each case of attrition, the treatment program continued with the remaining group members. When the study concluded, one subject was randomly deleted from each of two groups (groups 31 and 12) so that all treatment cells contained 16 persons.

Subjects' ages ranged from 16 to 62 with the mean age being 29.4 years. The somewhat high mean age was due primarily to the large non-student portion of the sample (30.2%). A more detailed demographic breakdown of the sample is given in Table 1 and Table 2. In Table 1 the demographic variables were tabulated according to cohort, in

Table 1

Demographic Data for 160 Anxious Subjects

Demographic Descriptors		Numbers of Persons in each Category						Summary
Variable	Categorization	Cohort 1	Cohort 2	Cohort 3	Cohort 4	Cohort 5	Total	%
Age:	20-	7	2	1	9	11	30	18.75
$\bar{x}$=29.4	21 - 24	7	14	4	9	7	41	25.63
	25 - 29	12	8	2	5	2	29	18.13
	30 - 39	7	5	3	9	9	33	20.63
	40 - 49	2	5	1	5	7	20	12.50
	50+	2	1	1	1	2	7	4.38
Sex	Male	15	13	4	13	11	56	34.60
	Female	22	22	8	25	27	104	65.40
Self assessed anxiety level	High	16	15	4	20	14	69	43.10
	Medium	20	18	8	17	21	84	52.90
	Low	1	2	0	1	3	7	3.90
Had received psychiatric care	Yes	8	9	4	7	9	36	23.00
	No	29	26	8	31	29	123	77.00
Occupation	Student	28	34	9	29	30	110	68.75
	Education	18	6	6	10	5	45	28.13
	Arts	3	4	0	6	3	16	10.00
	Science	1	2	0	8	6	17	10.63
	Engineering	1	2	0	0	0	3	1.88
	Other	5	10	3	5	6	29	18.13
	Non-Student	9	11	3	9	18	50	31.25
Referral source	Student Newspaper	22	20	5	12	5	64	37.80
	City Newspaper	0	1	1	10	16	28	16.50
	Television	4	8	2	0	0	14	8.10
	Radio	2	0	0	3	0	5	2.90
	Class Announcement	6	2	4	18	18	48	28.20
	Other	2	2	0	4	3	11	6.50

Table 2

Demographic Data for 160 Subjects Subdivided According to Treatment Condition

Demographic Descriptors		Numbers of Persons in each Treatment Group*											Summary
Variable	Categorization	11	12	21	22	31	32	41	42	51	52	Total	%
Age:	20-	5	3	3	3	1	4	2	4	1	2	30	18.75
	21 - 24	3	5	5	4	6	3	4	6	3	2	41	25.63
	25 - 29	3	2	3	0	6	4	1	1	4	5	29	18.13
	30 - 39	3	3	0	5	3	3	6	3	4	3	33	20.63
	40 - 49	2	3	2	2	0	1	2	2	3	3	20	12.50
	50+	0	0	1	2	0	1	1	0	1	1	7	4.38
	$\bar{x}$ (years)	27.3	28.9	26.8	32.0	26.2	28.2	31.4	26.9	32.4	31.7	29.4	-
Sex	Male	8	7	5	5	5	3	8	4	7	4	56	34.60
	Female	8	9	11	11	11	13	8	12	9	12	104	65.40
Self assessed anxiety level	High	8	6	5	7	6	9	7	5	8	8	69	43.10
	Medium	8	10	11	8	10	7	7	9	7	7	84	52.90
	Low	0	0	0	1	00	0	2	2	1	1	7	3.90
Had received psychiatric care	Yes	4	4	3	5	3	4	4	2	3	5	37	23.00
	No	12	12	13	11	13	12	12	14	13	11	123	77.00
Occupation	Student	12	11	14	10	12	11	10	14	8	8	110	68.75
	Education	5	4	4	6	7	5	3	5	4	2	45	28.13
	Arts	3	0	2	1	1	2	2	1	1	3	16	10.00
	Science	2	2	5	1	0	2	2	2	0	1	17	10.63
	Engineering	0	1	0	0	1	0	0	1	0	0	3	1.88
	Other	2	4	3	2	3	2	3	5	3	2	9	18.13
	Non-student	4	5	2	6	4	5	6	2	8	8	50	31.25
Referral source	Student Newspaper	7	4	8	5	8	7	5	9	5	6	64	37.60
	City Newspaper	2	3	12	2	3	1	3	3	3	6	28	16.50
	Television	0	2	1	2	2	3	0	0	1	3	14	8.10
	Class Announcement	4	6	6	5	4	6	6	5	2	4	48	28.20
	Other	1	0	1	2	2	2	2	0	1	0	11	6.50

* Treatment groups are numbered according to Figure 1, page 39.

Table 2 the results were tabulated according to treatment condition.

Equipment and Facilities

All sessions were carried out in a standard 10' x 16' lab. Subjects sat on comfortable lounge chairs (usually used for relaxation training), the experimenter sat in a chair facing the subjects, and the research assistant at a table adjacent to the experimenter. All EMG units were housed on book cases behind the subjects. The data acquisition equipment was on the table in front of the research assistant.

All EMG levels were measured using an Autogenics Systems Inc., 1700 electromyograph. Each subject had their own EMG. A one second response averaging mode was used in generating the feedback signal and a 100-200 Hz bandpass was used, as recommended by the manufacturer. The muscle tension information from all four subjects was processed simultaneously using the Autogenics System Inc. 5600 data acquisition center and printer assembly. Electrodes were applied in the standard manner for measuring frontal EMG (cf. Budzynski, 1973) and impedences of 10,000 ohms or less were maintained throughout.

Dependent Measures

Three dependent measures were used in this study: the IPAT Self-Analysis Form (IPAT) (Cattell, 1957), the State-Trait Anxiety Inventory (STAI) (Spielberger, 1968), and frontal EMG under baseline conditions. These measures are discussed in turn below.

IPAT Self-Analysis Form

The IPAT is a 40 item inventory intended to measure manifest anxiety level, whether it is situationally determined or relatively independent of the immediate situation (Cattell & Scheier, 1963). It has demonstrated adequate reliability (Guilford, 1959; Scheier, 1963) and validity (Cattell & Scheier, 1963; Cohen, 1965) and IPAT scores are reported to correlate highly with physiological measures of anxiety (Cohen, 1965; Smith, 1973).

State-Trait Anxiety Inventory

The State-Trait Anxiety Inventory (STAI) is a 40 item self-evaluation questionnaire designed to measure and distinguish between stable individual differences in anxiety proneness (A-Trait) and transitory anxiety reactiveness (A-State) (Spielberger, Gorsuch & Lushene, 1970). It is composed of 20 items that ask a subject to respond according to how he "generally feels" (A-Trait items), and 20 items that ask a subject to respond according to how he "feels right now" (A-State items). This scale has been used extensively in anxiety research (e.g., D'Augelli, 1974; Martuza, 1974; Spielberger, 1975; Townsend, *et al.*, 1975) and demonstrates adequate reliability (Kendall, Finch, Auerbach, Hooke & Mikeulka, 1976; Spielberger *et al.*, 1970) and validity (Kendal *et al.*, 1976; Martuza, 1974; Spielberger, 1975; Spielberger *et al.*, 1970).

EMG Level

EMG data was obtained in the manner usually adopted in biofeedback research (cf. Townsend *et al.*, 1975; Wilkinson, 1976). After attaching surface electrodes to the frontal region (two positive electrodes placed one inch above the eyebrow vertically above the center of the eye with a ground mid-way between) the subjects were told to relax as much as possible, using whatever strategy they usually used to relax. Subjects were given about 30 seconds to settle themselves, then EMG recording was begun. Ten second average microvolt levels were recorded with a 90 second average being output for the initial, middle and final 90 seconds of a five minute baseline period.

Treatment Procedures

Subjects were assigned to treatment conditions in a semi-random (read nonsystematic) fashion by the secretary for the project. The available times were read to the subject, who then chose from among the alternatives. Each treatment group began with a short explanation about the project in general, followed by administration of the STAI and IPAT. Following completion of questionnaires all subjects received a short presentation on the nature of anxiety which concluded with a discussion on muscle tension as a correlate of anxiety, the monitoring of muscle tension using an EMG, and a 5 minute EMG baseline period. This procedure required about 35 minutes of the first session. After the 5 minute EMG baseline subjects then began their respective treatment programs. These programs are outlined below.

Group 1: EMG Biofeedback Training

All subjects in EMG feedback conditions received six, 20 minute training sessions. Three 90 second probes of EMG activity were taken at the 2 minute, the 10 minute and the 20 minute point in each training session. All subjects received individual information about muscle tension levels via click feedback presented through headphones. In addition, all subjects were instructed to practice "duplicating the same feeling state associated with slow clicking" for 15-20 minutes at home every day between sessions. A short discussion about observations, awareness, strategies, etc., followed each training session.

In the first EMG training session (which occurred immediately after the assessment and introduction periods described above) subjects were instructed to focus their attention on the clicking and to explore a variety of strategies that might be successful in slowing the clicking rate. This exploration was to be conducted with an objective attitude focusing on the clicking, discarding strategies that didn't produce the desired effect and working with strategies that were accompanied by slower clicking. In addition, towards the end of the first session, subjects were instructed to do a mental scan through their bodies for any different feelings or sensations that might be associated with the slower clicking.

In session two the three stages in the biofeedback training process (cf. Budzynski, 1973) were explained and a discussion about the nature of passive control was developed. In the second training session, subjects were instructed to become more selective in their exploration, with the goal in mind of developing a strategy that would reliably produce and maintain a slower rate of clicking.

In the last four sessions emphasis was placed on identifying the personal ideosyncratic feeling indicators that could serve as internal feedback to tell the person when they were in a relaxed state. To give opportunity to practice controlling muscle tension in a variety of situations, subjects remained hooked up to the EMG and received feedback during the post training discussion period of each session. In session four the idea of a stimulus cue to elicit relaxation was introduced, and subjects were instructed to rehearse their cue several times at the end of the training session when the clicks became quite slow. In session five transfer considerations were introduced. Subjects received some periods where the volume was turned off while they produced or maintained relaxation, receiving a periodic return to feedback condition in order to check out their perception of being relaxed. In sessions five and six subjects received instruction in using their stimulus cue to enhance their ability to relax prior to anticipated stressors.

Group 2: Cognitive Monitoring Training

After the assessment and introductory portions of the initial session, subjects in group two received instruction in the use of Wolpe's (1969) notion of SUDS to cognitively self-monitor anxiety level. The analogy of developing a mental speedometer to measure anxiety level was used to help explain the process involved. After discussing this procedure for self-monitoring subjects were informed of the possible benefits of self-monitoring and the results of an earlier research project utilizing this treatment program (Hiebert, 1976). Subjects were then instructed to monitor their anxiety (i.e., SUDS) levels at one hour intervals recording their results on the pocket sized pads provided.

In session two discussion centered around difficulties encountered with the monitoring program and awareness gained from the systematic recording of anxiety level. A series of slides were then shown depicting a wide variety of different situations ranging with respect to the amount of anxiety that might be experience (e.g., walking in a park, driving on a mountainous road, seeing a lizard, standing on the top of a 40 storey office building). Subjects were instructed to project themselves into each situation and monitor their anxiety level. Frontal EMG was also recorded for subsequent correlational use. The order of the slide sequence was varied between groups to counteract interdependency effects in the correlations. For inter-session practice, subjects were instructed to monitor anxiety level at 30 minute intervals for 2 or 3 days, then to monitor at 15 minute intervals even though they continued to record their SUDS readings every 30 minutes. Subjects were told that the reason for this procedure was to develop the habit of attending to their anxiety level in an objective manner: typically people monitoring in this way begin to catch themselves earlier in the sequence of becoming anxious, which allows them the option of responding earlier in the arousal sequence, before the anxiety begins to interfere too drastically with their functioning. The use of the monitoring pads would gradually be faded out, until the person was monitoring anxiety level on a strictly cognitive level.

In session three any difficulties and awarenesses were again discussed, after which subjects were instructed to begin fading out the use of the monitoring pads.

In session four the monitoring pads were collected and checked to make sure the subjects had been doing the intersession practice. Subjects were then instructed to focus their attention on the coping mechanisms used after an increase in anxiety level was noticed.

In sessions five and six, group discussion focused on individual coping mechanisms used to reverse perceived rising anxiety levels, perceived benefits of the cognitive monitoring program, and ways to maintain the habit of systematically monitoring anxiety level.

Group 3: EMG Biofeedback Training Augmented with Cognitive Monitoring Training

This program was essentially a combination of the programs operating in groups one and two. The first session for group 3 was identical to the first session for group 1. In the second session, the notion of cognitive monitoring as a means for enhancing transfer in a biofeedback training program was introduced after the training period. Subjects were given monitoring pads and assigned the same intersession practice as subjects in group 2. Biofeedback training continued as outlined for group 1, and cognitive monitoring training continued as outlined for group 2.

Group 4: High Expectancy Discussion Group

After the assessment and introductory portions of the initial session, the notion that anxiety reactions stemmed from underlying personal dynamics was introduced in group 4. The idea was developed that often personal value systems and belief patterns, issues of self-autonomy and other directedness, and a person's sense of general well-being and self-directedness lay at the heart of anxiety reactions. The way to offer real, lasting relief from anxiety was to focus on these underlying causes, rather than merely the anxiety symptoms. In subsequent sessions, the discussion in group 4 focused on the "underlying causes" referred to above. Group interaction was promoted and an attempt was made to include each group member in the discussion.

Group 5: No Contact Control

The agenda for this group has been previously outlined above. Persons in this treatment condition were told that it was necessary to obtain a stable indication of anxiety level before treatment could begin. This would be done by having these subjects come into the lab on three occasions to fill out some questionnaires, and to relax for 5 minutes while a machine measured muscle tension levels. The dates of these assessments coincided with the assessment dates for the treatment groups. After the no contact condition was finished, these subjects were assigned to biofeedback practicum students for treatment.

Systematic Desensitization Conditions

After two sessions half of the subjects in each treatment condition began a systematic desensitization program directed at a person anxiety producing situation. The first two sessions were identical for desensitization and nondesensitization subjects and are outlined above. The desensitization procedure was identical for all subjects and was introduced in addition to the existing treatment program. In the first desensitization session (session 3), a counter conditioning model for substituting relaxation habit for anxiety habits was introduced. The steps in a desensitization program were then outlined and sample hierarchies were discussed. Subjects were then given index cards for use in constructing individual hierarchies as a homework assignment. This session concluded with a brief relaxation exercise (for groups 2 and 4) or an EMG training session (for groups 1 and 3).

Session four for all desensitization subjects began with the checking of individual hierarchies for possible sequencing or spacing difficulties. This was followed by a 20 minute EMG feedback training session for groups 1 and 3, and a short taped relaxation sequence for groups 2 and 4. Following the relaxation period taped instructions for the presentation of hierarchy scenes were given. Subjects were instructed to prepare the scenes they were going to work on that day prior to the relaxation period (while the EMG electrodes were being applied). During the scene presentation subjects were instructed to visualize each scene three times (times of 5, 10, and 20 seconds were used with a one minute relaxation period between scenes) and then to: (a) proceed to the next item if no increase in anxiety was experienced, or (b) remain on the same item if any anxiety was experienced. Subjects in groups 1 and 3 continued to receive feedback during the desensitization procedure. Subjects in groups 2 and 4 had EMG monitored during the desensitization procedure but received no EMG feedback.

The agenda for sessions five and six were essentially the same as for session four. Subjects began by preparing the mental images of the scenes they were going to work on during that session while the EMG electrodes were being applied. An EMG training session or a relaxation sequence followed, and the hierarchy presentation concluded the session. A commercially available relaxation tape, and self-densensitization tape was used for all sessions (Fitzsimmons, 1977).

In order to maintain equal therapist contact across all groups, all desensitization subjects were posttested during session six regardless of whether or not they had finished their hierarchies. Subjects not finishing their hierarchies were given the choice of completing the hierarchy at home, or arranging an additional session for that purpose.

Summary

This was basically a two stage, five group repeated measures design comparing changes in anxiety level accompanying EMG biofeedback training, cognitive monitoring training and systematic desensitization. The study utilized high expectancy discussion and waiting list control groups. Anxiety levels were measured by Spielberger's STAI (Spielberger, 1968), Cattell's IPAT (Cattell, 1957) and baseline frontal EMG. Subjects were self-diagnosed anxious persons ranging from age 16 to 62 ($\overline{X} = 29.4$) and encompassing a wide variety of work settings (two-thirds of the sample were students). The result of the study follow in Chapter Four.

CHAPTER IV

DATA ANALYSIS AND CONCLUSIONS

The number of treatment groups, repeated measures design, and number of dependent measures used in this study make data analysis difficult. Therefore an overview of the statistical procedures used and the general findings will be given first, to act as an advance organizer for the interpretation of the tables and figures appended to this paper.

Overview

Data were analyzed using a three-way analysis of variance according to Figure 2. The results of this analysis, for all dependent measures are presented in Appendix A. All treatment groups demonstrated significant anxiety decrements. However, the anxiety decreases were not equally consistent or reliable across all treatment groups for all dependent measures. Basically, treatment groups employing EMG biofeedback demonstrated more consistent anxiety reductions across all dependent measures. Subjects assigned to cognitive monitoring training and systematic desensitization conditions demonstrated significant anxiety decrements on some dependent measures. However, adding either cognitive monitoring training or systematic desensitization to EMG biofeedback did not produce a more powerful effect than using EMG biofeedback alone. Subjects in the high expectancy discussion group experienced anxiety reductions on the smallest number of dependent measures.

		Anxiety Level		
Treatment Stage		Pretest (T_1)	Posttest 1 (T_2)	Posttest 3 (T_3)
Treatment Mode Factor B	Factor A			
1. Training/ Monitoring	1. EMG biofeedback training			
	2. Cognitive monitoring training			
	3. Biofeedback + cognitive monitoring			
	4. High expectancy discussion group			
	5. Waiting list			
2. Systematic Desensiti-zation	1. EMG biofeedback training			
	2. Cognitive monitoring			
	3. Biofeedback + cognitive monitoring			
	4. High expectancy discussion group			
	5. Waiting list control			

Figure 2. Diagram for data analysis. Table entries were group mean scores. One set of data incorporated IPAT scores, another incorporated STAI-S scores, another STAI-T scores, and another EMG levels.

Data Analysis

The results of the data analysis are presented in more detail in the remainder of this chapter. First the research hypotheses listed in Chapter II will be discussed, afterwhich some analyses of related concerns will be presented. For the sake of clarity, the hypotheses will be dealt with sequentially, beginning with hypothesis #1.

Hypothesis #1

> Persons receiving treatment in the form of EMG biofeedback training or instruction in cognitive monitoring will experience an anxiety decrement, as evidenced by: (a) pretreatment-posttreatment score comparisons on all anxiety measures; (b) treatment-control (expectancy) posttreatment score comparison, and (c) treatment-control (no contact) posttreatment score comparisons.

Findings. The three-way Analyses of Variance referred to above depicted a significant times effect for all dependent measures: $F(2,300) = 44.6$, $p < .01$, for STAI-S scores; $F(2,300) = 76.36$, $p < .01$, for STAI-T scores; $F(2,300) = 39.61$, $p < .01$, for IPAT scores; and $F(2,300) = 27.02$, $p < .01$, for EMG levels. (The group means for STAI-S, STAI-T, IPAT scores and EMG levels are listed in Tables 3 to 6 and portrayed graphically in Figures 3-6. The summaries of Analysis of Variance appear in Appendix A.)

The above data suggest that on the average, subjects experienced a reduction in anxiety regardless of which treatment program they received. Significant interaction terms for some of the dependent

Table 3

Mean STAI-S Scores for 160 Subjects

Treatment		Times		
A	B	1	2	3
1	1	43.625	40.375	36.938
1	2	47.000	35.063	33.688
2	1	40.750	36.688	34.688
2	2	47.375	41.875	37.125
3	1	45.875	42.750	38.500
3	2	49.563	38.250	36.875
4	1	42.625	35.750	36.188
4	2	43.313	44.414	34.938
5	1	42.063	41.938	40.688
5	2	46.063	41.000	40.125

Table 4

Mean STAI-T Scores for 160 Subjects

Treatment		Times		
A	B	1	2	3
1	1	49.938	44.563	39.438
1	2	48.063	43.063	42.375
2	1	42.813	40.125	37.813
2	2	51.250	48.063	45.313
3	1	49.375	48.188	44.688
3	2	53.625	47.938	45.250
4	1	45.688	42.313	36.875
4	2	46.625	44.625	41.313
5	1	47.250	44.313	44.563
5	2	48.000	45.563	43.688

Table 5

Mean IPAT Scores for 160 Subjects

Treatment		Times		
A	B	1	2	3
1	1	41.188	37.500	32.875
1	2	39.563	38.750	35.875
2	1	35.938	32.188	29.000
2	2	43.750	41.125	34.000
3	1	43.438	40.438	34.250
3	2	48.063	42.563	39.938
4	1	34.375	31.625	25.500
4	2	38.875	39.625	37.750
5	1	39.000	39.813	38.938
5	2	41.625	43.750	42.500

Table 6

Mean 5 Minute EMG Baseline Levels for 160 Subjects

Treatment		Times		
A	B	1	2	3
1	1	1.720	1.495	1.200
1	2	2.221	1.730	1.426
2	1	2.147	1.878	1.415
2	2	2.397	2.291	2.247
3	1	1.898	2.056	1.433
3	2	2.445	1.689	1.357
4	1	2.001	1.858	1.737
4	2	1.535	1.577	1.440
5	1	2.158	2.157	1.968
5	2	1.915	2.083	1.872

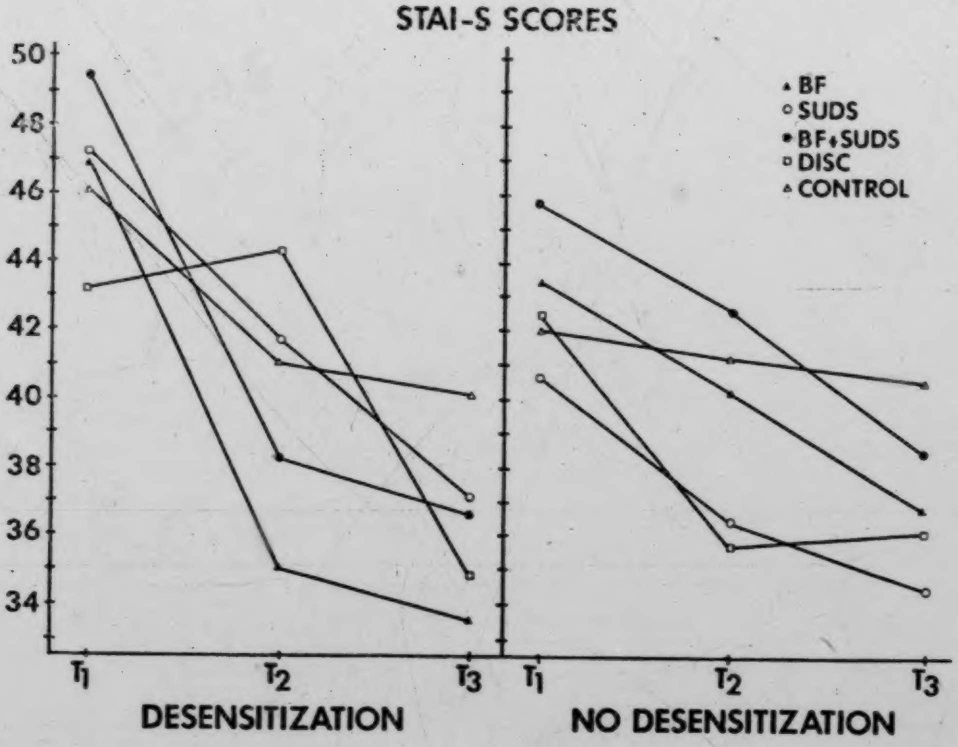

Figure 3. Mean STAI-S Scores for 160 Subjects

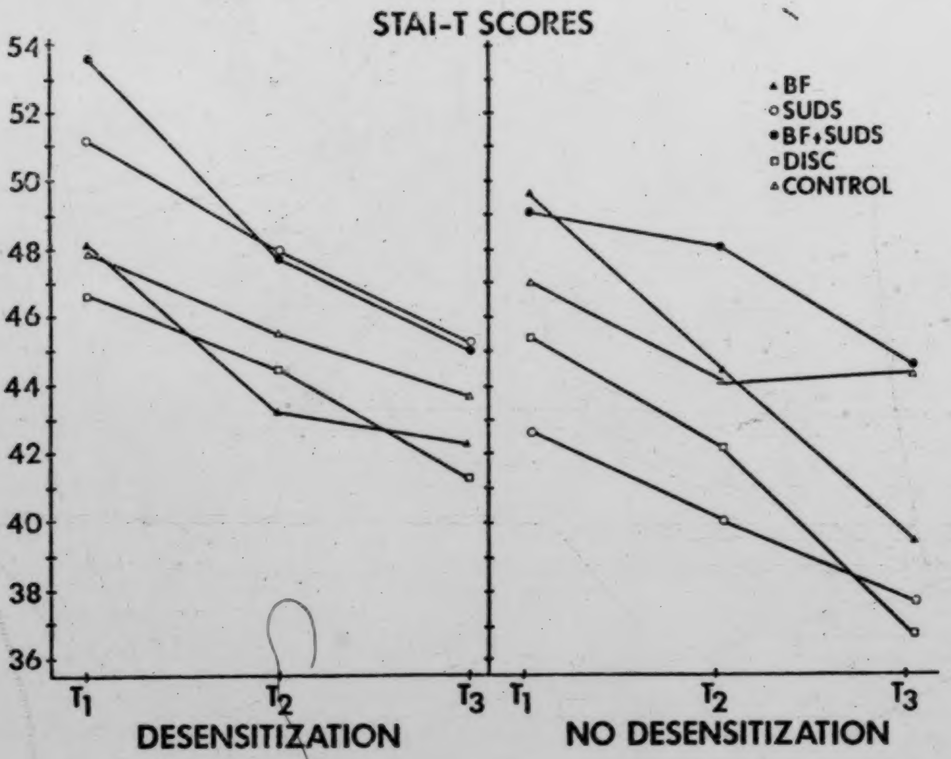

Figure 4. Mean STAI-T Scores for 160 Subjects.

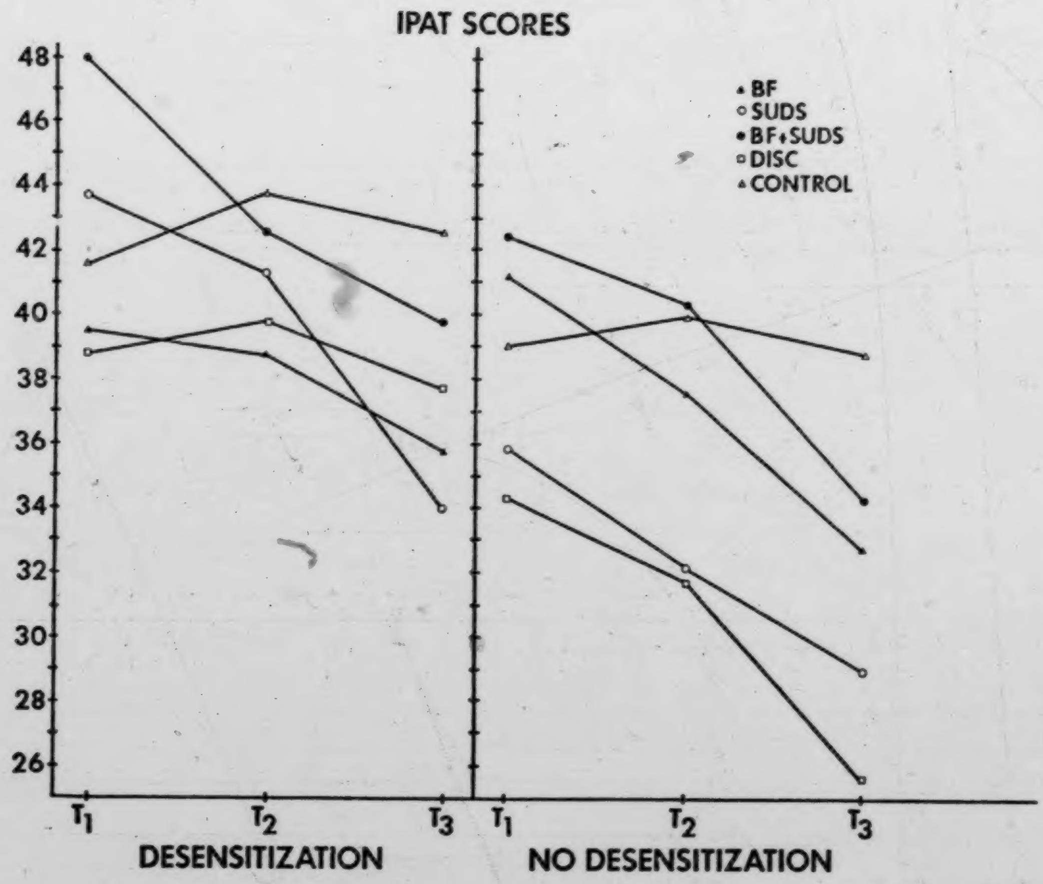

Figure 5. Mean IPAT Scores for 160 Subjects.

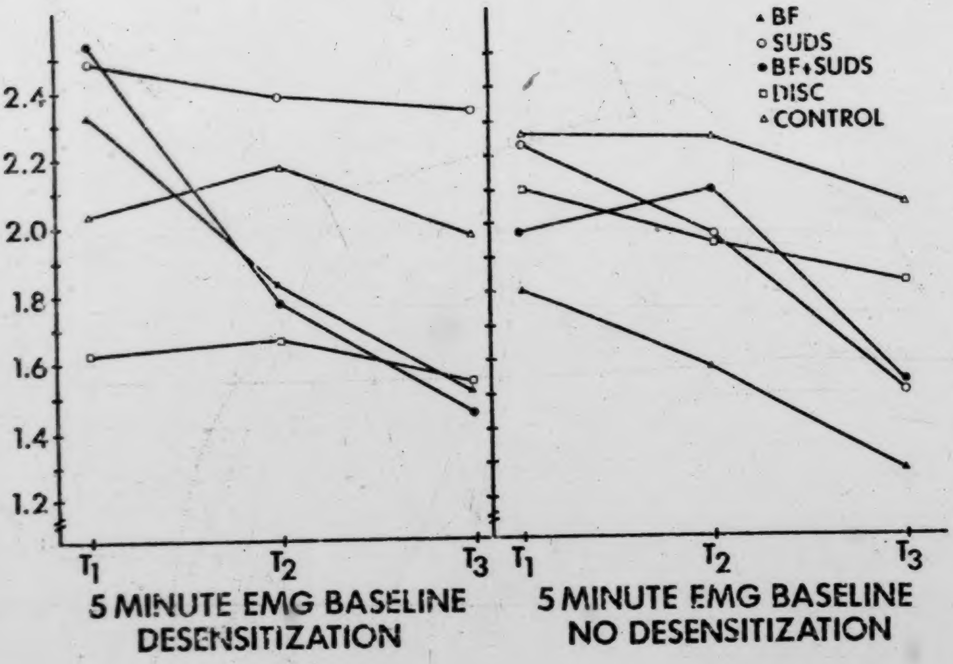

Figure 6. Mean Baseline EMG Levels for 160 Subjects.

measures suggests that the anxiety decrements are not uniform across all treatment groups. Specifically, the significant A x C interaction for IPAT scores, F(8,300) = 3.34, $p \leqslant .001$, indicates differential reduction in anxiety level, across time, for the five treatment groups. (The A x C IPAT means are listed in Table 7.) The Scheffé Multiple Comparison procedure was used to determine the critical anxiety decrement for A x C IPAT means ($\overline{X}_1 - \overline{X}_2 \geqslant 5.48$). Significant reductions in anxiety were found in groups 1, 2, and 3, but not in groups 4 or 5, Sch(8,300) $\geqslant$ 15.52, $p \leqslant .05$. (It should be noted that this procedure is identical to the tests for simple main effects discussed by Winer, 1962.) Scrutiny of the pretest scores in Table 7 revealed large differences in initial anxiety level, which could account for the posttest differences. An analysis of covariance using pretest scores as a covariate was conducted to address this concern. (The adjusted means from this analysis appear in Table 8 and the analysis of covariance appears in Appendix B.) Significant between group differences were found in the adjusted IPAT means, F(4,149) = 4.04, $p < .01$. Scheffé Multiple Comparisons indicated that adjusted IPAT means for groups 1, 2, and 3 were significantly lower than group 5, Sch(1,149) $\geqslant$ 3.91, $p < .05$.

A significant A x C interaction was also observed in the EMG data, F(8,300) = 2.49, $p \leqslant .01$. (The A x C EMG means are listed in Table 9). The results of Scheffé Multiple Comparisons revealed significant reduction in muscle tension levels only in groups 1 and 3, Sch(8,300) $\geqslant$ 15.68, $p < .05$.

Table 7

Group IPAT Means for A x C Interaction

Treatment Group	Times			Decrement*	
	1	2	3	T_1-T_2	T_1-T_3
1	40.38	38.13	34.38	2.25	6.00
2	39.84	36.66	31.50	3.18	8.34
3	45.25	41.50	37.09	3.75	8.16
4	36.63	35.63	31.63	1.00	5.00
5	40.31	41.78	40.72	-1.47	- .41

*Decrements greater than 5.48 are significant, $p < .05$.

Table 8

Adjusted Group IPAT Posttest Means for Factor A

Treatment Group	Unadjusted Mean	Adjusted Mean*
1	34.38	34.47
2	31.50	33.40
3	37.09	32.93
4	31.63	34.99
5	40.72	40.87

*Prestests scores (T_1) were used as the covariate.

Table 9

Group EMG Means for A x C Interaction

Treatment Group	Times 1	2	3	Decrement* $T_1 - T_2$	$T_1 - T_3$
1	1.971	1.612	1.313	.359	.658
2	2.272	2.084	1.831	.188	.441
3	2.172	1.872	1.395	.30	.777
4	1.768	1.717	1.588	.051	.180
5	2.036	2.120	1.920	.084	.116

*Decrements greater than .53 are significant, $p < .05$.

Conclusions. Hypothesis #1 is partially confirmed. Support was obtained for part (a) on all dependent measures. In part (b) significant anxiety decrements were evident for groups 1, 2 and 3. Significant decrements in IPAT scores and EMG levels were evident in groups 1 and 3, but no significant decrements were observed in group 4 on either of these measures. Part (c) received support from the EMG data where significant between group differences were found.

Hypothesis #2

The anxiety reduction hypothesized in #1 will be retained over time, as evidenced by the self-report of a random sampling of subjects one month after the termination of treatment.

Findings. A random sampling of subjects from each cohort was contacted by telephone about one month after posttesting. The experimenter explained that a follow-up contact was being made and asked two questions: (a) "Have you noticed any changes in your anxiety since the research project finished?" and (b) "Was it your initial impression that the program helped reduce your anxiety or did it really not help that much?" The responses to these questions are reported in Table 10. These results were anlysed using a Chi square test of independence. A significant relationship was found between the initial impression of treatment effectiveness and the maintenance of the treatment effect, $\chi^2 = 3.79$, $p < .05$. When the results are analysed according to perceived benefit of treatment one month following treatment termination 73.9% of the subjects in the

Table 10

Summary of Follow-up Responses

Initial Impression	Treatment Effect		
	Maintained	Not Maintained	Total
Yes it helped	29	5	34
No, not really	7	5	12
TOTAL	36	10	46

follow-up sample reported substantially less anxiety.

Conclusion. Hypothesis #2 is substantiated.

Hypothesis #3

> EMG biofeedback training and instruction in cognitive monitoring will be equally effective in producing an anxiety decrement, as evidenced by inter-group comparison of posttreatment anxiety level.

Findings. The results of the Scheffé Multiple Comparisons referred to above indicate that subjects receiving EMG biofeedback training, groups 1 and 3, experienced significant reductions in muscle tension levels, while the reduction demonstrated by the cognitive monitoring group, group 2, was not significant. No differences were found on any of the other dependent measures.

Conclusions. Hypothesis #3 must be rejected. Although no intergroup differences were found, a significant reduction in muscle tension was evident in groups 1 and 3 but not in group 2. This would suggest that EMG biofeedback training is more efficacious in producing muscle tension decrements than is cognitive monitoring training.

Hypothesis #4

> People who receive biofeedback training within the cognitive framework provided by cognitive monitoring training will experience a greater anxiety reduction than people who receive either biofeedback training or instruction in cognitive

monitoring in isolation, as evidenced by intergroup comparison of posttreatment anxiety level.

Findings. As reported earlier, there were no significant between group differences in the anxiety decreases demonstrated by groups 1, 2 and 3. Significant muscle tension decrements were observed in groups 1 and 3, but not in group 2 (see Table 9).

Conclusions. As a whole, hypothesis #4 must be rejected. Support was obtained for concluding that the combination of cognitive monitoring training and biofeedback training was more effective than cognitive monitoring training alone. The combined treatment was not significantly more effective than EMG biofeedback training alone.

Hypothesis #5

Persons who receive systematic desensitization as an adjunct to biofeedback training or cognitive monitoring training will experience a greater anxiety reduction than persons who receive biofeedback training or cognitive monitoring training in isolation, as evidenced by inter-group comparisons of posttreatment anxiety level.

Findings. The Summary of Analysis of Variance given in Appendix A shows a significant difference between the IPAT score of subjects receiving desensitization and those not receiving desensitization, $F(1,150) = 6.76$, $p \leq .01$. (Appropriate group means are given in Table 11.) Specifically, subjects in desensitization groups had higher initial IPAT scores, a condition which remained throughout

Table 11

Group IPAT Means for Factor B Across Time

Treatment Group	Times 1	 2	 3
1	38.59	36.31	32.11
2	42.38	41.16	38.01

treatment. Analysis of Covariance confirmed that posttest differences in IPAT scores were unlikely to be the result of pretest differences $\underline{F}(1,149) = 4.72$, $\underline{p} \leq .03$. (See Appendix B for the Summary of Analysis of Covariance and Table 12 for the adjusted means.)

Greater anxiety decrements in nondesensitization conditions were also reflected by the significant B x C interaction in the STAI-S data, $\underline{F}(2,300) = 3.77$, $\underline{p} \leq .03$. (See Table 13 for the relevant group means.) Using the Scheffé Multiple Comparison procedure, the critical anxiety decrement was found to be 4.63 for scores in the same row, $\underline{Sch}(2,300) \geq 6.06$, $\underline{p} < .05$, and 2.98 for scores in the same column, $\underline{Sch}(1,450) \geq 3.84$, $\underline{p} < .05$. Both the nondesensitization and desensitization groups demonstrated significant decrements in STAI-S scores during the course of treatment. Initially (T_1), STAI-S scores were significantly higher for desensitization subjects, but no significant between group differences were evident in posttest scores (T_3). However, it should be noted that this effect is due primarily to the significant anxiety decrement which occurred between T_1 and T_2, prior to commencing the desensitization program. A more clear demonstration of the role of desensitization was obtained by analysing the significant A x B x C interaction, $\underline{F}(8,300) = 2.26$, $\underline{p} < .03$. (See Table 6 for the appropriate means.) The Scheffé procedure mentioned earlier was used to determine the magnitude of the EMG decrement necessary to attain significance. Mean differences greater than .465 were found to be significant, $\underline{Sch}(2,300) \geq 6.06$, $\underline{p} < .05$. (Mean differences are given in Table 14.) Scrutiny of Table 6 indicates no significant reduction in EMG levels for groups receiving

Table 12

Adjusted Group IPAT Posttest Means for Factor B

Treatment Group	Unadjusted Means	Adjusted Means*
1	32.11	33.77
2	38.01	36.90

*Pretest scores (T_1) were used as covariate.

Table 13

Group STAI-S Means for B x C Interaction

Treatment Groups	Times		
	1	2	3
1	43.0	39.5	37.4
2	46.7	40.1	36.6

Table 14

Group EMG Means for A x B x C Interaction

Treatment		Decrement*		
A	B	T_1-T_2	T_2-T_3	T_1-T_3
1	1	.225	.295	.520
1	2	.491	.304	.795
2	1	.296	.463	.732
2	2	.106	.044	.150
3	1	-.158	.623	.465
3	2	.756	.332	1.088
4	1	.143	.121	.264
4	2	-.042	.137	.095
5	1	.001	.189	.190
5	2	-.168	.211	.043

*Decrements greater than .46 are significant, $p < .05$.

desensitization (groups 12, 22, and 32) during the time when they were receiving desensitization (i.e., between T_2 and T_3). Groups 12 and 32 demonstrated a significant muscle tension reduction prior to the introduction of desensitization. The EMG decrement for group 22 was not significant. Alternatively, all of the nondesensitization groups (groups 11, 21 and 31) experienced significant reductions in muscle tension.

Conclusions. Hypothesis #5 must be rejected. In this study systematic desensitization seemed to have a nonfacilitative effect on other treatment procedures.

Hypothesis #6

> People who receive systematic desensitization as an adjunct to biofeedback training will experience a greater anxiety reduction than persons who receive traditional systematic desensitization, as evidenced by inter-group comparison of posttreatment anxiety level.

Findings. The results of the Scheffé Multiple Comparisons described above suggest that EMG augmented desensitization (groups 12 and 32) is a more effective treatment than traditional desensitization (group 42). Subjects in group 42 did not demonstrate a significant EMG decrement, whereas subjects in groups 12 and 32 did exhibit significant decreases in frontal EMG.

Conclusions. Hypothesis #6 is supported. However, a word of caution is in order. The higher initial EMG levels in groups 12 and

32 would lead some to argue that intergroup differences are largely responsible for the significant results reported above. These intergroup differences in baseline EMG also raise a question concerning the interval nature of EMG data. It is possible that the decrement of .225 μv in group 11 is comparable to the decrement of .491 μv in group 12 considering the baseline differences (1.720 μv versus 2.221 μv). Clearly more research is necessary before a categorical claim can be made.

Additional Analyses

Therapist Effects

In this study the experimenter acted as the therapist for all treatment groups. A question immediately arises concerning the extent to which differential therapist expectancies might account for variations in treatment effectiveness. In order to determine therapist equivalence and perceived treatment effectiveness across groups, all subjects were asked to rate therapist characteristics (competence, likeableness, understanding, genuineness, etc.), and perception of treatment program (meaningfulness, effectiveness, etc.), at the conclusion of treatment (cf. Shaw, 1976). This rating scale was administered by the research assistant with the therapist absent. (A copy of this scale is given in Appendix C.) A Kruskal-Wallis one way analysis of variance by ranks indicated no significant differences between groups on either of these dimensions. (See Appendix D.) Therefore it seems unlikely that differences in anxiety decreases across groups is a function of therapist variables.

Additional support for the above claim was discovered after analysing the treatment data collected for wait list controls. When the project was finished approximately 20 people were still on the waiting list and interested in receiving treatment. To accommodate these people, two graduate student biofeedback therapists were hired to conduct EMG training programs. These two therapists were briefed in the experimental and data collection procedures for the study and assigned case loads from waiting list.

Upon completion of treatment, two, two-way analyses of variance for repeated measures were conducted to determine the equivalence of therapist functioning: a 3(therapist) x 3(repeats) ANOVA was conducted on the treatment data (see Table 15 for the groups means and Appendix E for the Analysis of Variance and Analysis of Covariance) and a 3(therapist) x 5(training session) ANOVA was conducted on EMG readings during training. All subjects where complete data were available were included in this analysis. Although all subjects received six training sessions, complete data were only available for five of the sessions due to equipment failure. No significant therapist differences were found in any of the ANOVAs. The analysis of covariance on the treatment means indicated a significant therapist effect for STAI-T scores, $\underline{F}(2.28) = 4.96$, $\underline{p} < .02$. Scheffé multiple comparisons indicated that a significant difference in adjusted posttest scores between therapist 1 (the experimenter) and therapist 3, $\underline{Sch}(2.28) = 7.57$, $\underline{p} \leq .05$. No other between therapist differences were significant. Therefore, the majority of evidence would support the position that subjects benefitted equally from treatment regardless

Table 15

Mean Pretest, Posttest Means on all Self-Report Measures for Three Different Therapist Groups

Dependent Measure	Therapist	Test Time		Adjusted Posttest
		Pretest	Posttest	
STAI-S	Th 1	43.63	36.94	36.75
	Th 2	42.90	34.30	34.44
	Th 3	42.67	38.50	38.75
STAI-T	Th 1	49.94	39.44	37.60
	Th 2	46.50	42.10	43.24
	Th 3	44.33	42.00	45.01
IPAT	Th 1	41.19	32.88	31.73
	Th 2	37.80	34.60	36.55
	Th 3	40.17	39.33	39.12

of which therapist they interacted with.

Variation across Cohorts

Procedurally, this study utilized four replications of the same design. The question arises as to whether or not similar results were obtained for all cohorts. As part of the data analysis presented above, a 3-way analysis of variance for repeated measures was performed on the data from each cohort as well as from the total sample. As can be seen from the tables in Appendix F, the pattern depicted in the total group data is essentially the same as that reflected in the data for each cohort.

Intercorrelations among Dependent Measures

This study used four dependent measures. It is of interest to investigate the intercorrelations between the dependent measures. The correlation matrix of pretest scores appears in Table 16. As can be seen, there is a significant correlation between the self-report measures. Of interest also is the higher correlation between the STAI-S and the STAI-T, and the lower correlation between the STAI-S and the IPAT.

The IPAT was the only self-report measure to correlate significantly with the initial EMG baseline measure.

Demographic Analysis: Dependent Measures

Post hoc scrutiny of the data suggested that treatment outcome may have been related to some demographic variables. To test this suspicion several two-way analyses of variance for repeated measures (demographic variable x time) were conducted. Those variables where

Table 16

Relationship Between Dependent Measures

Correlations Matrix

Variable	STAI-S	STAI-T	IPAT	EMG
STAI-S	1.000	0.564	0.481	0.101
STAI-T	0.564	1.000	0.728	0.129
IPAT	0.481	0.728	1.000	1.000
EMG	0.101	0.129	0.194	1.000

Probabilities of T

Variable	STAI-S	STAI-T	IPAT	EMG
STAI-S	0.000	0.000	0.000	0.203
STAI-T	0.000	0.000	0.000	0.105
IPAT	0.000	0.000	0.000	0.014
EMG	0.203	0.105	0.014	0.000

Degrees of Freedom = 158

significant differences were observed are reported below.

Age. Significant differences were observed in muscle tension decrements across time for different age categories. (See Table 17 for mean EMG levels and Appendix G for the Summary of Analysis of Variance.) These differences were not reflected in any of the self-report measures.

Scheffé Multiple Comparisons indicated that significant EMG decrements were experienced in the 25 years and younger, in the 30-39 years and the 50 years and over age categories, Sch(2,308) $\geqslant$ 6.06, $p < .05$. Significant differences were also evident in initial baseline EMG levels. The 30-39 years and the 50 years and over age categories had significantly higher EMG levels than those whose age was 29 years and younger, Sch(5,462) $\geqslant$ 11.15, $p < .05$. Upon completion of treatment, significant differences in EMG level were still observed between 30-39 year olds and those being 25 years and younger, Sch(5,462) $\geqslant$ 11.15, $p < .05$; other differences were no longer significant.

Generally speaking, younger subjects (29 and under) demonstrated lower muscle tension than older subjects (ages 30-39 and 50 or more). Additionally, younger subjects (25 and under), older subjects (50 and over) and subjects aged 30-39 demonstrated greater muscle tension decrements during treatment than subjects ages 25-29 and 40-49.

Sex. Significant male-female differences were observed in IPAT scores and EMG levels. (See Table 18 for the means and Appendix H for the Analysis of Variance and Covariance). The analysis of

Table 17

Mean EMG Levels for Age (Factor A)
Across Time (Factor B)

Age	Test Time 1	2	3
20 -	1.863	1.467	1.296
21 - 24	1.734	1.481	1.188
25 - 29	1.693	1.702	1.486
30 - 39	2.662	2.551	2.164
40 - 49	2.011	2.255	1.908
50 +	2.865	2.117	2.021

Table 18

Mean Scores for Sex (Factor A)
Across Time (Factor B)

Variable	Sex	Test Time 1	2	3
IPAT	male	37.37	35.40	32.88
	female	42.20	40.58	37.24
EMG	male	1.760	1.530	1.284
	female	2.201	2.076	1.790

covariance produced no significant differences in IPAT posttest scores covaried over pretest, however, there were still significant differences in adjusted posttest EMG levels, $\underline{F}(1,157) = 7.34$, $\underline{p} < .01$.

From the above analysis it would appear that male subjects not only had lower EMG levels than female subjects, but that males experienced a greater decrease in muscle tension levels during the course of treatment.

Self-assessed anxiety level. As part of the demographic questionnaire administered in session 1 subjects were asked to rate their anxiety level on a three point scale: high, medium or low. To ascertain the extent to which treatment outcome covaried with perceived anxiousness, self-assessed anxiety level was entered as one factor (having three levels: H, M, L) on a two-way analysis of variance for repeated measures. A significant group effect was found for STAI-T scores, $\underline{F}(2,157) = 7.55$, $\underline{p} < .01$. (See Table 19 for the mean STAI-T scores and Appendix I for the Summary of Analysis of Variance.) This would indicate that subjects accurately described their anxiety level as being high or medium or low. The resulting significant group effect could be attributed to differential treatment effectiveness between groups or to pretest differences (the group x times interaction was nonsignificant). To test this proposition an Analysis of Covariance was conducted, with pretest scores as the covariate (see Appendix I for the Analysis of Covariance). No significant differences were found in the adjusted posttest scores, indicating that the significant group effect in the Analysis of Variance was probably due to pretest differences.

Table 19

Mean Scores for Self-Assessed Anxiety Level (Factor A) Across Time (Factor B)

Variable	Self-Assessed Anxiety Level	Test Time 1	Test Time 2	Test Time 3
STAI-T	High	52.74	49.77	45.92
	Medium	45.58	41.76	39.62
	Low	40.13	38.00	37.88
IPAT	High	44.52	42.95	39.30
	Medium	38.12	36.27	32.97
	Low	32.63	30.50	28.00

Analysis of Biofeedback Training Data

It was postulated that presenting biofeedback within a cognitive monitoring framework (treatment group 3) would facilitate learning to self-regulate muscle tension. To test this suspicion two analyses of variance for repeated measures were conducted, one for the first 90 seconds in each training session (beginning) and another for the final 90 seconds in each training session (end). The mean EMG levels are presented in Table 20. The Summaries of Analysis of Variance are presented in Appendix J. A significant times effect was observed in both cases, $F(5,310) = 8.59$, $p < .01$ for beginning scores, $F(5,310) = 4.98$, $p < .01$ for end scores. Scheffé multiple comparisons indicated that the significant decrease in beginning EMG scores was attributed largely to the EMG (no cognitive monitoring) group (group 1). The significant interaction in the EMG end scores, $F(5,310) = 2.24$, $p < .05$, suggested that the muscle tension decreases were not consistent for both groups. Scheffé multiple comparisons indicated that the EMG decrements had attained significance by the fourth training session. The EMG decrement between sessions one and six for group 3 was not significant. The above data would suggest that presenting EMG biofeedback training within the cognitive monitoring framework has a deleterious effect on the rate of skill acquisition.

Demographic Analysis: Training Data

Post hoc scrutiny of the EMG data obtained at the beginning and end of each training session suggested that further analysis of the data according to demographic categories was appropriate. In each case two way analyses of variance for repeated measures were conducted

Table 20

Mean EMG Level for Six Training Sessions

Probe	Group	Training Session 1	2	3	4	5	6
Beginning	1	1.760	1.727	1.587	1.437	1.430	1.235
	3	1.700	1.747	1.675	1.761	1.405	1.415
End	1	1.682	1.420	1.306	1.162	1.133	1.196
	2	1.467	1.317	1.280	1.437	1.262	1.308

on the EMG beginning and EMG end data. The significant results are reported below.

Age. Mean EMG levels for the beginning and end of each training session are presented in Table 21. The summaries of Analysis of Variance appear in Appendix K. A significant between groups effect, $F(4,59) = 4.1$, $p < .01$ and groups x times interaction, $F(20,295) = 2.33$, $p < .01$, was observed in the beginning scores. Scheffé results indicated that subjects in the 30-39 year old category had higher muscle tension levels, $Sch(4,354) \geq 9.6$, $p < .05$, and experienced less reduction in muscle tension, $Sch(5,295) \geq 11.2$, $p < .05$, than subjects who were older or younger. This effect was mainly due to the pattern of scores for the 30-39 year old category: beginning muscle tension levels increased in sessions 2, 3 and 4 before returning to initial levels in sessions 5 and 6. Although beginning EMG levels in session 1 were highest in the 40 years and older category, this group demonstrated a significant reduction in beginning EMG levels over the six training sessions, $Sch(5,295) \geq 11.2$, $p < .05$.

A significant between groups effect, $F(4,59) = 4.11$, $p < .01$, and groups x times interaction, $F(20,295) = 1.59$, $p < .05$ was also observed in the EMG end scores. This effect was due mainly to differences between the 20 years and younger and the 40 years and older age groups, $Sch(4,354) \geq 9.48$, $p < .05$. Again subjects 40 years and older demonstrated significant decreases in EMG end levels, as did subjects ages 21-24, $Sch(5,295) \geq 11.25$, $p < .05$.

Thus it would appear that 30-39 year old subjects had higher muscle tension at the beginning of the training session and that the

Table 21

Mean EMG Levels for Age Groups for Six Training Sessions

Probe	Age Range	Training Sessions 1	2	3	4	5	6
Beginning	20 -	1.583	1.417	1.363	1.179	1.208	1.128
	21 - 24	1.756	1.535	1.360	1.467	1.300	1.159
	25 - 29	1.493	1.575	1.367	1.367	1.238	1.287
	30 - 39	1.753	2.595	2.440	2.448	1.763	1.782
	40 +	2.309	2.072	2.191	2.126	1.944	1.583
End	20 -	1.336	1.060	0.964	1.061	0.984	1.030
	21 - 24	1.634	1.333	1.179	1.134	1.112	1.106
	25 - 29	1.361	1.264	1.325	1.222	1.172	1.248
	30 - 39	1.845	1.641	1.453	2.020	1.414	1.873
	40 +	1.987	1.899	1.894	1.427	1.567	1.299

progression of scores is most eratic in this age group (see Table 21). Further, 21-24 year old subjects, and subjects 40 years and older demonstrated the largest reductions in EMG levels during the course of the six training sessions.

Sex. Mean EMG levels for the beginning and end of each training session are presented in Table 22. The summaries of Analysis of Variance are given in Appendix L. A significant between groups effect was observed in both the beginning EMG scores, $F(1,62) = 16.14$, $p < .01$, and the end EMG scores, $F(1,62) = 16.58$, $p < .01$. Specifically, the EMG levels for male subjects were lower both in the beginning and at the end of each training session. Both male and female subjects demonstrated significant decreases in beginning and ending EMG levels across the six training sessions.

Initial Baseline Scores

After baseline EMG readings are obtained the subject invariably inquires as to how their readings compare to those of other subjects. Answers to such questions are difficult because of the dearth of normative information. This author is unaware of any published EMG baseline norms. Even though 160 subjects is not a large norming sample it was deemed appropriate to compile the EMG baseline data in such a manner so as to permit normative comparisons with subjects possessing similar demographic characteristics to the subjects in this study. Appendix M contains histograms of raw frontal EMG levels obtained from a five minute baseline. The data is presented in Table 23 along with the appropriate deciles. It is hoped that this

Table 22

Mean EMG Levels for Males and Females for Six Training Sessions

Probe	Sex	Training Sessions 1	2	3	4	5	6
Beginning	Male	1.368	1.305	1.132	1.044	1.009	0.9731
	Female	1.933	1.979	1.911	1.910	1.647	1.523
End	Male	1.268	1.032	0.922	0.884	0.857	0.929
	Female	1.746	1.557	1.462	1.533	1.388	1.433

Table 23

Five Minute Frontal EMG Baselines on 160 Self-Diagnosed Anxious Subjects

Decile	Raw Scores Male	 Female	 Total Sample
1	.892 & below	1.555 & below	1.036 & below
2	.893 - 1.036	1.156 - 1.333	1.037 - 1.282
3	1.037 - 1.306	1.334 - 1.454	1.283 - 1.432
4	1.307 - 1.412	1.455 - 1.694	1.433 - 1.622
5	1.413 - 1.629	1.695 - 1.931	1.623 - 1.774
6	1.630 - 1.758	1.932 - 2.124	1.775 - 2.044
7	1.759 - 1.861	2.125 - 2.371	2.045 - 2.325
8	1.862 - 2.284	2.372 - 2.758	2.326 - 2.742
9	2.285 - 3.050	2.759 - 3.342	2.743 - 3.298
10	3.051 & over	3.343 & over	3.299 & over
Mean	1.75	2.20	2.04
Standard Deviation	.75	1.09	1.00

initial effort may promote the sharing of such data by other researchers.

Additional Findings

The question sometimes arises concerning the existence of therapeutic strategies that will facilitate rapid change. It will be recalled that the first two test times in this study were only one week apart. During that week the primary emphasis in all treatment conditions was on awareness. In some groups there was a systematic approach to training awareness via EMG biofeedback training and/or cognitive monitoring training. In another group the approach was less structured. In order to determine the differential effects these approaches might have on anxiety level a 5(treatments) x 2(times) 2-way analysis of variance for repeated measures was conducted. The group means are presented in Table 24 and the summary of analysis of variance is given in Appendix N. Significant reductions in IPAT scores were observed in groups 2 and 3, Sch(1,155) > 3.91, $p < .05$, and group 1, Sch(1,155) > 3.52, $p < .06$. This result would suggest that a systematic approach to awareness of anxiety is more effective in producing rapid anxiety decrements than discussion oriented approaches.

Summary

The data and relevant conclusions have been presented above. In general, anxiety treatment programs incorporating EMG feedback were found to be more effective than programs not having EMG biofeedback as a component. In fact combining other procedures with EMG biofeedback training was seen to have a deleterious effect in some cases.

Table 24

Mean IPAT Scores During Awareness Stage of Treatment

Group	Test Time	
	T_1	T_2
1	40.38	38.13
2	39.84	36.66
3	45.25	41.50
4	36.63	35.63
5	40.31	41.78

Differential treatment effects were observed for certain demographic variables. Age was found to be a factor influencing both treatment outcome, and degree of skill acquisition in EMG biofeedback training. Sex was also found to have differential effects: males demonstrated lower anxiety levels and lower muscle tension than did females.

It should be noted that the conservative nature of the Scheffé procedure might tend to increase type II error. However, it could also be argued that using a conservative test increases confidence in the results: significant differences resulting from a stringent test would be shown to have an even smaller probability of occurring by chance if a less conservative procedure had been employed.

Some discussion of these results and the implications arising from this study will be discussed in Chapter V. Additionally, some directions for future research in this area will be offered.

CHAPTER V

DISCUSSION AND IMPLICATIONS

Summary of Results

It would appear that EMG feedback focusing on awareness and control of muscle tension is more effective in producing anxiety decreases than some traditional procedures. Combining traditional anxiety treatment procedures with EMG biofeedback is unlikely to enhance the anxiety decrements obtained by using EMG biofeedback alone. Using EMG biofeedback to develop increased awareness of changes in muscle tension levels and to cultivate a skill in willfully relaxing muscle tension appears to be sufficient to produce significant anxiety decreases without the use of adjunctive procedures. Most subjects in this study were able to apply these skills to specific environmental stressors without a specific program aimed at substituting relaxation habits for anxiety habits. In this study, the inclusion of a specific habit substitution program (desensitization) in the therapeutic format did not have a facilitative effect. The initial work of Hiebert (1976) in cognitive monitoring training was replicated with similar results. Discussing basic life issues that are part of a person's anxiety experience was accompanied by anxiety reduction on some dependent measures. This alludes to the role of life style change in long term anxiety reduction. These issues are discussed in more detail below.

Clinical Significance

In research of this nature concern often arises over the possible distinction between statistically significant results and clinically significant results. Basically the question is, "Regardless of the statistical results, how much did people change during treatment?" Some representative examples are given below to illustrate the type of clinical changes that were observed.

Generally speaking, the most dramatic changes were observed in group four. One female subject (J) decided to confront her supervisor and change jobs, both of which she had contemplated for years. A marked change in J's appearance was also observed, specifically her face took on a more youthful appearance. The change was so marked that the relief research assistant (who had been present on day one but who had not met with the group again until day six) failed to recognize J on the final day of treatment. A substantial reduction in fidget behavior was observed in another male subject. This subject also demonstrated a large reduction in muscle tension during the course of treatment. Another male subject (A) was certain that no change in his anxiety could be accomplished in only three weeks. By the third week, he revised his statement to say that the change certainly would not be maintained. In the follow-up contact, A expressed surprise and pleasure that the anxiety relief had been maintained. Two subjects in a later cohert were friends of A and had volunteered primarily because of A's recommendation.

Substantial change was also observed in other treatment groups. M, a female subject in group three reported that just monitoring her

SUDS levels resulted in her staying more relaxed, even in situations that she usually found quite upsetting. An older female subject in group one reported being less irritable with her husband—more tolerant and feeling less hassled. A male subject in group one reported the disappearance of a tension headache during the fourth training session. A male subject in group two reported feeling less pressure at work and recommended our study to three of his neighbors.

Generally speaking, the self-reports of subjects emphasized an increased sense of well-being and control over their anxiety. This is not to say that all subjects experienced profound anxiety relief. A snake phobic subject B, in group three reported that desensitization was not working at all. Upon closer questioning B stated that she didn't really want to be relaxed around snakes, she only volunteered to take part in the study because her husband wanted her to. A salesman in group one was convinced that his fear of addressing groups of people was too severe and too long-standing to be altered much in three weeks. He reported no therapeutic benefit from treatment. The above examples are typical representations of people reporting no therapeutic benefit in this study.

To summarize, there was generally speaking, not only a statistically significant effect but substantial clinical change as well. The number of subjects recommending friends and relatives to the study can be taken as an indication of the perceived effectiveness of the treatment procedures. The practice of recommending friends was observed in all treatment conditions which further attests to the perceived equivalence of the therapeutic effectiveness of all treatment programs.

Limitations and Possible Solutions

In most research studies certain problems arise that place limitations on the conclusions drawn. Some of the most prominent concerns are discussed below and alternative procedures are suggested where appropriate.

Generalizability

In any study investigating the effectiveness of clinical treatment procedures, the extent to which the research conclusions can be generalized to a clinical setting is an important consideration. The broad cross-section of subjects, the wide range of ages, the type of furniture and physical arrangements in the lab all enhance the appropriateness of generalizing the results to a clinical context. However, some limitations do exist. Subjects were randomly assigned to treatment groups, a condition that almost never exists in a clinical setting. Random group assignment resulted in some subjects receiving a treatment that was not maximally appropriate, e.g., some subects experiencing diffuse anxiety were assigned to desensitization conditions and experienced difficulty identifying a theme for hierarchy construction.

This difficulty could be avoided by establishing definite criteria for matching client problems to therapeutic treatment procedure. An independent therapist could assign subjects to treatment groups on the basis of these criteria and a detailed history conducted at a pretreatment assessment session. Such procedures have begun to be used in research of this nature (Hardt, Note 4).

In this study muscle relaxation was used to inhibit an anxious response. A problem arises when one onsiders that many people do not react to anxiety inducing stimuli with muscle tension increases. Other common parameters of physiological reactivity include: peripheral vasoconstriction, heartrate and respiration increases and changes in GSR. It is reasonable to deduce that a person who reacts to environmental stressors with peripheral vasoconstriction may not experience much anxiety-inhibitory effect from muscle tension training. Recently some therapists (cf. Budzynski, Note 1) have used a stress profile to determine a person's pattern of physiological reactivity. This is accomplished by monitoring several different physiological parameters while the person experiences a variety of standard stressors. Biofeedback training usually follows utilizing the person's most reactive physiological parameter. Thus a person who reacts with muscle tension increases will commence an EMG biofeedback program, while a person who responds with peripheral vasoconstriction will embark on a hand temperature biofeedback program. In this way a person learns to self-regulate his own idiosyncratic physiological anxiety response. To this writer, the procedure outlined above has more logical appeal than training muscle relaxation and assuming that it will have an anxiety-inhibitory effect for all people.

Care was taken to ensure that the treatment procedures used in this study closely approximated those used by practicing clinicians. The physical setting and the broad sample used in this study enhance the extent to which the results of this study are useful to clinicians.

However, the above concerns should be kept in mind when applying the results of this research to clinical situations.

Procedural Concerns

The content and step-by-step procedure for each treatment program were carefully planned and tested in pilot work. In spite of these precautions some procedural difficulties were encountered which may have affected treatment outcome. Probably the most salient area for concern lies with the amount of material covered during the course of treatment. The pace of the sessions was not rushed, but each minute was necessary in order to cover the agenda for that session. The therapists assisting in the completion of treatment for persons on the waiting list at the end of the program reported difficulty in remaining within the time lines that existed in the study. Typically the sessions lasted 10 to 15 minutes longer with these therapists. A possible reason for this larger time requirement may have been that the other two therapists had less experience with biofeedback than the author, however, it is also possible that too much information was packed into some sessions. Certainly longer sessions would have been necessary without a research assistant to help in the application of electrodes and the recording of data.

The design of this study stipulated equal numbers of sessions for all subjects in all treatment programs. This was necessary in order to control for therapist interaction time. However, controlling for therapist interaction time created other problems. Some subjects did not learn control over muscle tension within the six treatment sessions. Some desensitization subjects did not finish their hierarchies by the sixth session. Subjects not completing hierarchies were given the opportunity to receive extra sessions in order to finish, however, all subjects were posttested during session six regardless of whether or not they had demonstrated control of EMG or completed their hierarchies.

In the future, it might be appropriate to follow some criterion oriented guidelines cast within certain maximum therapist-interaction time constraints. For example EMG biofeedback subjects could train to a criterion of 2.0 μv or a 50% reduction in baseline frontal muscle tension or 10 training sessions, whichever occurred first. Desensitization subjects could complete their hierarchies or receive five desensitization sessions whichever occurred first. Subjects would be posttested upon the attainment of the criterion, rather than at a set time interval. This procedure would more closely follow a clinical practice than predetermining the number of treatment sessions for all subjects.

Another procedural problem was encountered by subjects not keeping their initial appointment. In all cases, subjects who missed the first session were not allowed to commence treatment in the second or subsequent sessions and the treatment group continued with fewer than four subjects. This could have been avoided by having all subjects complete a pretest and history taking one week prior to treatment onset. An adequate supply of subjects would then be the only condition necessary to reduce "no shows" to minimal proportions. If the volunteer rate experienced in this study is typical, the adequate supply of subjects would not be a matter for concern.

In spite of the above procedural difficulties, it was possible to maintain a high degree of similarity in content, sequence, and rate of presentation across cohorts and across groups sharing common treatment procedures. In fact the research assistants expressed surprise at how closely the agendas were followed and how consistently the treatment programs were conducted across cohorts.

A Note on Systematic Desensitization

The effectiveness of desensitization within this treatment context remains somewhat surprising. Basically, EMG augmented desensitization was more effective than traditional desensitization, but not any more effective than EMG biofeedback alone. The combination of EMG biofeedback and systematic desensitization in this study is really best viewed as EMG augmented desensitization rather than a combination of two treatment programs. Therefore the question still remains as to whether or not adding desensitization to a biofeedback program enhances the treatment effect, i.e., given that subjects learn to willfully control muscle tension levels and are instructed in procedures for transferring that skill outside the laboratory setting and applying the skill to certain idiosyncratic stresses, can these subjects bring that skill to bear on specific anxiety habits themselves, or is it more efficacious to use a systematic habit substitution program to accomplish this effect? This remains an important clinical question to be addressed.

Future Directions

Miller (1978) suggests that the final stages in the validation of clinical procedures include controlled comparisons with the best available other treatment techniques, and broad clinical trials with large samples under the conditions to be expected in general use. Bronfenbrenner (1976) and Goldman (1976) both advocate the ultimate testing of clinical procedures under conditions that closely approximate clinical settings. With these considerations in mind, it would be possible to devise a sequel to the study presented in this thesis that would follow these general guidelines and incorproate some of the suggestions made in the earlier part of this chapter. Such a study might commence by conducting an individual history and a stress profile on all subjects. This could be followed by the administration of pretests and a short tapeslide sequence explaining the nature of anxiety, and the role of biofeedback in anxiety treatment. Subjects would then be assigned to treatment group by the intake assistant on the basis of pre-established clinical criteria. This intake procedure would occur approximately one week before treatment commenced to allow scheduling of treatment groups. The group format worked well in the present study and could be retained in the sequel as a means of attaining a larger sample.

In order to investigate the appropriateness of biofeedback training in the physiologically deviant modality a screening procedure like the one mentioned above would permit the investigation of several client-treatment variables. It would be possible to address the appropriateness of utilizing biofeedback training based on the stress profile by having half of the subjects train on their most physiologically reactive modality while the other half experienced an EMG training program. All subjects would train to pre-established criteria similar to those mentioned earlier in this chapter. Subjects who could identify an anxiety focus (as opposed to a pervasive feeling of anxiety) would be assigned to phobic condition. Subjects experiencing more defuse anxiety would be assigned to a nonphobic condition. Half of the phobic subjects would receive systematic desensitization after training criterion levels had been met, the other half would enter a follow-up condition. Upon concluding treatment subjects would be randomly assigned to one of three follow-up conditions. Subjects of follow-up in condition 1 would complete follow-up assessment one month, three months and eight months after treatment. In follow-up condition 2, subjects would complete follow-up assessment three months and eight months after treatment. Subjects in follow-up condition 3 would complete follow-up assessment eight months after treatment. Figure 7 gives a graphic representation of this procedure.

Such a design would compensate for many of the limitations of the present study. The group assignment procedures would increase the chances of appropriate treatment while maintaining minimal therapist bias in group composition. The establishment of training criteria would permit more valid conclusions to be drawn concerning treatment efficacy. The manner in which desensitization is introduced in this design would permit statements to be made concerning the efficacy of using biofeedback and desensitization in tandem, as well as

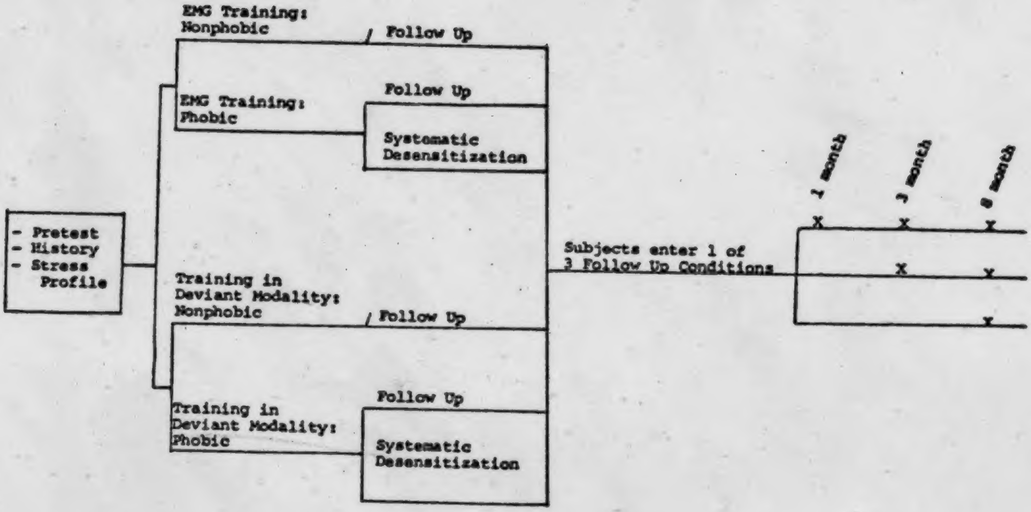
- Pretest
- History
- Stress Profile
EMG Training: Nonphobic
Follow Up
EMG Training: Phobic
Follow Up
Systematic Desensitization
Training in Deviant Modality: Nonphobic
Follow Up
Training in Deviant Modality: Phobic
Follow Up
Systematic Desensitization
Subjects enter 1 of 3 Follow Up Conditions
1 month
3 month
6 month
X
X
X
X
X
X

Figure 7. Diagram of Treatment Procedures for a Sequel Study

investigating the efficacy of using desensitization with nonmuscle-tension biofeedback modes. The follow-up procedures would shed light on the effectiveness of differing follow-up schedules.

This type of study would no doubt enhance the strength of the conclusions that could be drawn concerning the efficacy of biofeedback training and alternative anxiety treatment procedures. It would be possible to conduct the study, or portions of the study in a variety of clinical settings, using a variety of therapists. Such a practice would also add strength to the results. Such an undertaking would be expensive in terms of equipment costs, client hours and therapist time. Furthermore, the cooperation necessary for such an undertaking might be difficult to obtain. However, research in these or similar directions would undoubtedly enhance the knowledge pool in the clinical applications of biofeedback training.

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APPENDICES

APPENDIX A

SUMMARIES OF ANALYSES OF VARIANCE FOR THREE-WAY ANOVA'S FOR REPEATED MEASURES

Summary of Analysis of Variance (STAI-S)

Source	Sum of Squares	Degrees of Freedom	Mean Squares	F	p
Between Subjects	37970.81	159			
A	668.63	4	167.6	0.69	0.60
B	156.38	1	156.38	0.64	0.42
AB	659.50	4	164.88	0.68	0.61
Subjects within groups	36486.31	150	243.24		
Within Subjects	23886.69	320			
C	5058.88	2	2529.44	44.62	0.00
AC	589.00	8	73.62	1.30	0.24
BC	427.00	2	213.50	3.77	0.024
ABC	805.88	8	100.73	1.78	0.081
C x Subjects within groups	17006.88	300	56.69		

Summary of Analysis of Variance (STAI-T)

Source	Sum of Squares	Degrees of Freedom	Mean Squares	F	p
Between Subjects	44095.44	159			
A	1491.06	4	372.77	1.37	0.25
B	723.00	1	723.00	2.65	0.10
AB	1013.69	4	253.42	0.93	0.45
Subjects within groups	40867.69	150	272.45		
Within Subjects	9476.00	320			
C	3018.62	2	1509.31	76.36	0.00
AC	246.75	8	30.84	1.56	0.14
BC	18.38	2	9.19	0.46	0.63
ABC	263.81	8	32.98	1.67	0.10
C x Subjects within groups	5929.38	300	19.76		

Summary of Analysis of Variance (IPAT)

Source	Sum of Squares	Degrees of Freedom	Mean Squares	F	p
Between Subjects	69570.31	159			
A	3348.81	4	837.20	2.01	0.09
B	2818.06	1	2818.06	6.76	0.01
AB	850.1	4	212.53	0.51	0.73
Subjects within groups	62553.31	150	417.02		
Within Subjects	13014.69	320			
C	2448.56	2	1224.28	39.61	0.00
AC	825.13	8	103.14	3.34	0.00
BC	88.94	2	44.47	1.44	0.24
ABC	380.44	8	47.55	1.54	0.14
C x Subjects within groups	9272.50	300	30.91		

Summary of Analysis of Variance (EMG)

Source	Sum of Squares	Degrees of Freedom	Mean Squares	F	p
Between Subjects	326.25	159			
A	14.38	4	3.59	1.80	.13
B	.651	1	.651	0.33	.57
AB	11.17	4	2.79	1.40	.24
Subjects within groups	300.05	150	2.00		
Within Subjects	112.31	320			
C	15.42	2	7.71	27.02	.00
AC	5.68	8	.71	2.49	.01
BC	.45	2	.23	0.79	.45
ABC	5.17	8	.65	2.26	.02
C x Subjects within groups	85.60	300	.29		

APPENDIX B

TWO-WAY ANALYSIS OF COVARIANCE FOR IPAT SCORES

Summary of Analysis of Covariance

Source	SS	df	MS	F	p
A	1307.02	4	326.75	4.04	.004
B	381.16	1	381.16	4.72	.031
A x B	340.78	4	85.20	1.05	.381
Error	12042.41	149	80.82		

APPENDIX C

FEEDBACK FORM

FEEDBACK FORM

Rate each of the following characteristics of the research director (Bryan) on a 5 point scale from 0 (not at all) to 4 (very much so).

likableness	0	1	2	3	4
competence	0	1	2	3	4
ability to relate to people	0	1	2	3	4
knowledge of the area	0	1	2	3	4
ability to explain concepts	0	1	2	3	4
flexibility	0	1	2	3	4
efficiency	0	1	2	3	4
trustworthiness	0	1	2	3	4
consistency	0	1	2	3	4
credibility	0	1	2	3	4

Rate each of the following statements about your treatment group on a 5 point scale from 0 (not at all) to 4 (very much so).

sessions were meaningful	0	1	2	3	4
sessions were relevant	0	1	2	3	4
sessions were helpful	0	1	2	3	4
the program was effective	0	1	2	3	4

Do you feel less anxious now than you did when the program first began? ____________

Comments: ____

____

____

____

____

APPENDIX D

ANALYSIS OF FEEDBACK FORM

Kruskal-Wallis Analysis of Variance

Variable	Sums of Ranks							
	Treatment Condition							
	11	12	21	22	31	32	41	42
Therapist	1099.50	1099.50	1222.50	913.00	862.00	1279.50	1082.50	956.50
Treatment group	1164.00	1111.50	1146.50	1202.00	993.00	913.00	1000.00	985.00

Analysis of Variance

Variable	DF	H	Corrected H	P
Therapist	7.00	6.931	7.000	.45
Treatment group	7.00	3.540	3.620	.80

APPENDIX E

ANALYSIS OF VARIANCE AND COVARIANCE FOR THREE DIFFERENT THERAPIST GROUPS (THERAPIST x TIME)

Summary of Analysis of Variance of STAI-S Scores

Source of Variation	SS	DF	MS	F	p
Between Subjects	3736.75	31			
'A' Main Effects	41.62	2	20.81	0.163	0.85
Subjects within groups	3693.25	29	127.35		
Within Subjects	1969.00	32			
'B' Main Effects	574.89	1	574.89	14.027	0.00
'A*B' Interaction	45.04	2	22.52	0.559	0.58
'B' x Subject within groups	1189.58	29	41.01		

Summary of Analysis of Variance of STAI-T Scores

Source of Variation	SS	DF	MS	F	p
Between Subjects	5686.94	31			
'A' Main Effects	22.96	2	11.48	0.06	0.94
Subjects within Groups	5666.81	29	195.41		
Within Subjects	1464.00	32			
'B' Main Effects	451.32	1	451.32	27.91	0.00
'A*B' Interaction	164.09	2	82.04	5.07	0.01
'B' x Subject within Groups	468.88	29	16.17		

Summary of Analysis of Variance of IPAT Scores

Source of Variation	SS	DF	MS	F	p
Between Subjects	10237.94	31			
'A' Main Effects	125.67	2	62.84	0.180	0.84
Subjects within Groups	10139.44	29	349.64		
Within Subjects	1461.00	32			
'B' Main Effects	231.52	1	231.52	7.852	0.01
'A*B' Interaction	133.15	2	66.57	2.258	0.12
'B' x Subject within Groups	855.06	29	29.49		

Summary of Analysis of Covariance for STAI-S Scores

Homogeneity of Regression

Source	DF	SS	MS	F	p
Diff.	2	47.164	23.58	.405	.671
Error	26	1512.266	48.16		

Analysis of Covariance

Source	DF	SS	Adj F	p
Grp.	2	36.69	.659	.525
With	26	55.69		

Summary of Analysis of Covariance for STAI-T Scores

Homogeneity of Regression

Source	DF	SS	MS	F	p
Diff.	2	83.560	41.780	1.358	.275
Error	28	799.897	30.765		

Analysis of Covariance

Source	DF	MS	F	p
Grp.	2	156.560	4.962	.014
With	28	31.552		

Summary of Analysis of Covariance for IPAT Scores

Homogeneity of Regression

Source	DF	SS	MS	F	p
Diff.	2	159.698	79.849	1.374	.271
Error	28	1510.098	58.115		

Analysis of Covariance

Source	DF	MS	F	p
Grp.	2	145.252	2.434	.106
With	28	59.667		

APPENDIX F

MEANS AND ANALYSES OF VARIANCE FOR ALL COHORTS ACROSS ALL DEPENDENT MEASURES

Cohort 1: STAI-S Scores

Cell Means

A	B	1	2	3
1	1	45.75	41.25	35.75
1	2	46.00	32.25	27.50
2	1	35.75	41.25	37.50
2	2	43.25	33.50	30.00
3	1	39.75	40.50	36.00
3	2	47.75	36.25	35.00
4	1	42.25	35.75	33.50
4	2	32.25	33.00	27.00
5	1	44.50	40.75	40.50
5	2	41.50	39.25	39.00

Summary of Analysis of Variance

Source	SS	DF	MS	F	P
Between Subjects	4997.25	39			
A	657.38	4	164.34	1.29	0.30
B	297.69	1	297.69	2.33	0.14
AB	211.13	4	52.78	0.41	0.80
Subjects within Groups	3831.06	30	127.70		
Within Subjects	4275.38	80			
C	1197.06	2	598.53	16.72	0.00
AC	302.81	8	37.85	1.06	0.40
BC	205.38	2	102.69	2.87	0.06
ABC	421.94	8	52.74	1.47	0.19
C x Subjects within Groups	2148.19	60	35.80		

Cohort 1: STAI-T Scores

Cell Means

A	B	1	2	3
1	1	46.75	40.50	39.50
1	2	44.25	41.75	44.25
2	1	44.50	47.50	45.00
2	2	47.50	40.75	38.50
3	1	48.00	46.75	43.25
3	2	52.25	46.25	41.50
4	1	47.25	42.25	37.25
4	2	43.25	43.50	39.75
5	1	46.25	43.75	46.50
5	2	41.75	41.75	42.50

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	6149.31	39			
A	237.88	4	59.47	0.31	0.87
B	32.06	1	32.06	0.17	0.67
AB	122.38	4	30.59	0.16	0.96
Subjects within Groups	5757.00	30	191.90		
Within Subjects	1862.69	80			
C	389.813	2	194.91	11.81	0.00
AC	211.56	8	26.45	1.60	0.14
BC	1.81	2	0.91	0.05	0.95
ABC	269.00	8	33.63	2.04	0.06
C x Subjects within Groups	990.50	60	16.51		

Cohort 1: IPAT Scores

Cell Means

A	B	1	2	3
1	1	39.25	36.25	33.75
1	2	36.25	37.25	35.00
2	1	43.00	41.00	39.75
2	2	42.00	41.25	34.50
3	1	45.00	41.00	36.25
3	2	48.25	38.00	32.25
4	1	37.50	33.75	27.75
4	2	41.25	41.50	39.75
5	1	39.00	38.50	35.50
5	2	36.50	35.00	35.00

Summary of Analysis of Variance

Source	SS	DF	MS	F	P
Between Subjects	10031.19	39			
A	376.13	4	94.03	0.31	0.87
B	5.63	1	5.63	0.02	0.89
AB	424.44	4	106.11	0.35	0.85
Subjects within Groups	9225.00	30	307.50		
Within Subjects	2600.69	80			
C	690.44	2	345.22	14.21	0.00
AC	257.56	8	32.20	1.33	0.25
BC	1.88	2	0.94	0.04	0.96
ABC	193.31	8	24.16	0.99	0.45
C x Subjects within Groups	1457.50	60	24.29		

Cohort 1: EMG Baseline Levels

Cell Means

A	B	1	2	3
1	1	1.334	1.497	1.033
1	2	2.809	2.235	1.646
2	1	1.733	1.317	1.651
2	2	2.800	2.128	2.098
3	1	1.919	1.528	1.257
3	2	1.665	1.894	1.281
4	1	2.477	2.410	2.414
4	2	1.441	1.452	1.369
5	1	1.436	1.718	1.762
5	2	1.442	1.649	1.643

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	83.474	39			
A	2.813	4	0.703	0.32	0.861
B	0.570	1	0.570	0.26	0.613
AB	14.550	4	3.638	1.66	0.184
Subjects within Groups	65.541	30	2.185		
Within Subjects	15.244	80			
C	1.696	2	0.848	6.06	0.004
AC	3.476	8	0.435	3.10	0.005
BC	0.380	2	0.190	1.36	0.265
ABC	1.288	8	0.161	1.15	0.344
C x Subjects within Groups	8.404	60	0.140		

Cohort 2: STAI-S Scores

Cell Means

A	B	1	2	3
1	1	46.00	50.25	46.75
1	2	46.50	33.50	34.50
2	1	48.25	44.00	43.00
2	2	55.00	46.75	42.00
3	1	46.50	49.75	42.00
3	2	50.00	35.25	36.25
4	1	37.50	31.00	42.75
4	2	47.75	51.00	37.75
5	1	47.50	40.50	40.00
5	2	40.00	35.75	39.75

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	15962.56	39			
A	505.63	4	126.41	0.27	0.90
B	76.88	1	76.88	0.16	0.69
AB	1229.06	4	307.27	0.65	0.63
Subjects within Groups	14151.00	30	471.70		
Within Subjects	6606.69	80			
C	804.25	2	402.13	5.71	0.01
AC	180.38	8	22.55	0.32	0.96
BC	301.50	2	150.75	2.14	0.13
ABC	1091.56	8	136.45	1.94	0.07
C x Subjects within Groups	4229.00	60	70.48		

Cohort 2: STAI-T Scores

Cell Means

A	B	1	2	3
1	1	54.75	49.25	44.75
1	2	53.25	43.00	38.75
2	1	42.25	41.50	39.00
2	2	47.50	49.25	44.25
3	1	52.75	53.25	50.00
3	2	52.50	46.00	44.75
4	1	40.50	35.75	36.25
4	2	51.75	46.75	42.75
5	1	47.00	42.00	37.50
5	2	38.25	39.25	37.25

Summary of Analysis of Variance

Source	SS	DF	MS	F	P
Between Subjects	11589.25	39			
A	1440.44	4	360.11	1.19	0.33
B	10.19	1	10.19	0.03	0.86
AB	1089.38	4	272.34	0.90	0.47
Subjects within Groups	9049.25	30	301.64		
Within Subjects	2696.69	80			
C	852.44	2	426.22	18.16	0.00
AC	242.44	8	30.30	1.29	0.27
BC	6.75	2	3.38	0.14	0.87
ABC	187.06	8	23.38	1.00	0.44
C x Subjects within Groups	1408.00	60	23.47		

Cohort 2: IPAT Scores

Cell Means

A	B	1	2	3
1	1	43.75	44.75	41.00
1	2	39.00	39.00	34.00
2	1	30.00	29.75	30.25
2	2	40.00	36.50	32.75
3	1	44.00	44.75	36.50
3	2	47.50	49.50	45.50
4	1	33.25	29.75	25.75
4	2	40.50	39.25	39.25
5	1	41.50	40.75	37.75
5	2	38.75	44.75	42.50

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	16830.31	39			
A	3149.25	4	537.31	1.20	0.33
B	407.00	1	407.00	0.91	0.35
AB	876.63	4	219.16	0.49	0.74
Subjects within Groups	13397.44	30	446.58		
Within Subjects	1992.69	80			
C	294.88	2	147.44	6.46	0.00
AC	124.75	8	15.59	0.68	0.70
BC	18.44	2	9.22	0.40	0.67
ABC	184.81	8	23.10	1.01	0.44
C x Subjects within Groups	1369.81	60	22.83		

Cohort 2: EMG Baseline Levels

Cell Means

A	B	1	2	3
1	1	2.248	1.461	1.240
1	2	2.377	1.441	1.262
2	1	1.968	2.006	1.358
2	2	2.035	1.944	2.141
3	1	1.997	3.395	2.038
3	2	3.092	1.612	1.454
4	1	1.496	2.011	1.701
4	2	1.622	1.807	1.284
5	1	2.284	1.990	1.903
5	2	2.208	1.966	1.943

Summary of Analysis of Variance

Source	SS	DF	MS	F	P
Between Subjects	90.583	39			
A	6.425	4	1.606	0.58	0.676
B	0.110	1	0.110	0.04	0.843
AB	1.557	4	0.389	0.14	0.965
Subjects within Groups	82.490	30	2.750		
Within Subjects	45.312	80			
C	5.182	2	2.591	6.01	0.004
AC	4.736	8	0.592	1.37	0.227
BC	2.370	2	1.185	2.75	0.072
ABC	7.154	8	0.894	2.07	0.053
C x Subjects within Groups	25.870	60	0.431		

Cohort 3: STAI-S Scores

Cell Means

A	B	1	2	3
1	1	43.75	35.24	31.75
1	2	49.50	38.00	39.75
2	1	41.75	30.75	28.75
2	2	42.75	43.50	36.50
3	1	58.00	41.00	38.25
3	2	53.50	46.00	41.75
4	1	43.50	38.00	33.25
4	2	42.75	38.75	38.25
5	1	31.75	44.50	35.25
5	2	51.00	38.50	42.75

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	6534.25	39			
A	1144.25	4	286.06	1.87	0.14
B	612.13	1	612.13	4.00	0.05
AB	191.94	4	47.98	0.31	0.87
Subjects within Groups	4585.94	30	152.86		
Within Subjects	7170.00	80			
C	1779.31	2	889.66	13.62	0.00
AC	525.56	8	65.70	1.01	0.44
BC	56.31	2	28.16	0.43	0.65
ABC	889.00	8	111.13	1.70	0.12
C x Subjects within Groups	3919.81	60	65.33		

Cohort 3: STAI-T Scores

Cell Means

A	B	1	2	3
1	1	50.25	44.00	35.00
1	2	46.00	43.00	41.50
2	1	41.75	35.25	32.75
2	2	53.50	50.25	45.50
3	1	52.00	51.25	47.25
3	2	61.00	57.75	52.50
4	1	40.00	38.50	34.00
4	2	43.75	41.25	41.25
5	1	43.25	42.25	43.50
5	2	50.75	43.50	40.00

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	10161.19	39			
A	2610.06	4	652.52	3.22	0.03
B	864.06	1	864.06	4.26	0.05
AB	608.56	4	152.14	0.75	0.57
Subjects within Groups	6078.50	30	202.61		
Within Subjects	2561.38	80			
C	952.38	2	476.19	23.63	0.00
AC	108.63	8	13.58	0.67	0.71
BC	3.31	2	1.66	0.08	0.92
ABC	288.06	8	36.01	1.79	0.10
C x Subjects within Groups	1209.00	60	20.15		

Cohort 3: IPAT Scores

Cell Means

A	B	1	2	3
1	1	38.25	32.25	24.00
1	2	38.25	37.75	35.25
2	1	33.25	25.50	20.50
2	2	42.50	36.25	32.75
3	1	44.00	47.00	40.25
3	2	50.25	45.00	40.50
4	1	24.00	24.00	18.75
4	2	34.75	37.75	35.00
5	1	29.00	32.50	32.25
5	2	38.25	42.50	41.75

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	21462.50	39			
A	3301.00	4	825.25	1.59	0.20
B	2017.19	1	2017.19	3.88	0.06
AB	534.81	4	133.70	0.26	0.90
Subjects within Groups	15609.50	30	520.32		
Within Subjects	3817.38	80			
C	580.88	2	290.44	7.22	0.00
AC	584.75	8	73.09	1.82	0.09
BC	44.56	2	22.28	0.55	0.58
ABC	194.69	8	24.34	0.61	0.77
C x Subjects within Groups	2412.50	60	40.21		

Cohort 3: EMG Baseline Levels

Cell Means

A	B	1	2	3
1	1	2.005	1.599	1.480
1	2	1.820	1.488	1.243
2	1	1.678	1.617	1.350
2	2	2.402	2.377	2.094
3	1	1.662	1.891	1.302
3	2	2.036	1.569	1.046
4	1	2.023	1.428	1.233
4	2	1.712	1.770	1.524
5	1	2.280	2.410	2.123
5	2	2.318	2.831	2.182

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	49.937	39			
A	10.601	4	2.650	2.37	0.075
B	0.725	1	0.725	0.65	0.427
AB	3.048	4	0.762	0.68	0.610
Subjects within Groups	33.563	30	1.119		
Within Subjects	22.753	80			
C	4.197	2	2.098	7.99	0.001
AC	1.491	8	0.186	0.71	0.682
BC	5.737	2	2.869	0.11	0.897
ABC	1.261	8	0.158	0.60	0.773
C x Subjects within Groups	15.747	60	0.262		

Cohort 4: STAI-S Scores

Cell Means

A	B	1	2	3
1	1	39.00	34.75	33.50
1	2	46.00	36.50	33.00
2	1	37.25	30.75	29.50
2	2	48.50	43.75	40.00
3	1	39.25	39.75	37.75
3	2	47.00	35.50	34.50
4	1	47.25	38.25	35.25
4	2	50.50	54.50	36.75
5	1	44.50	42.00	47.00
5	2	51.75	50.50	39.00

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	8895.75	39			
A	1363.44	4	340.86	1.61	0.20
B	691.25	1	691.25	3.27	0.08
AB	493.25	4	123.31	0.58	0.68
Subjects within Groups	6347.81	30	211.59		
Within Subjects	5834.69	80			
C	1438.06	2	719.03	13.16	0.00
AC	278.94	8	34.87	0.64	0.74
BC	338.69	2	169.34	3.10	0.05
ABC	501.81	8	62.73	1.15	0.35
C x Subjects within Groups	3277.19	60	54.62		

Cohort 4: STAI-T Scores

Cell Means

A	B	1	2	3
1	1	48.00	44.50	38.50
1	2	48.75	44.50	45.00
2	1	42.75	36.25	34.50
2	2	56.50	52.00	53.00
3	1	44.75	41.50	38.25
3	2	48.75	41.75	42.25
4	1	55.00	52.75	40.00
4	2	47.75	47.00	41.50
5	1	52.50	49.25	50.75
5	2	61.25	57.75	55.00

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	15502.13	39			
A	1871.75	4	467.94	1.21	0.33
B	720.31	1	720.31	1.86	0.18
AB	1292.44	4	323.11	0.83	0.51
Subjects within Groups	11617.63	30	387.25		
Within Subjects	2355.38	80			
C	911.50	2	455.75	27.10	0.00
AC	229.75	8	28.72	1.71	0.12
BC	63.38	2	31.69	1.88	0.16
ABC	141.88	8	17.73	1.05	0.41
C x Subjects within Groups	1008.88	60	16.81		

Cohort 4: IPAT Scores

Cell Means

A	B	1	2	3
1	1	43.50	36.75	32.75
1	2	44.75	41.00	39.25
2	1	37.50	32.50	25.50
2	2	50.50	50.50	46.75
3	1	36.75	29.00	24.00
3	2	46.25	37.75	41.50
4	1	42.75	39.00	29.75
4	2	39.00	40.00	37.00
5	1	46.50	47.50	50.25
5	2	53.00	52.75	50.75

Summary of Analysis of Variance

Between Subjects	19226.06	39			
A	2901.06	4	725.27	1.62	0.20
B	1817.50	1	1817.50	4.06	0.05
AB	1064.13	4	266.03	0.59	0.67
Subjects within Groups	13443.38	30	448.11		
Within Subjects	3199.38	80			
C	795.25	2	397.63	14.22	0.00
AC	374.00	8	46.75	1.67	0.12
BC	142.00	2	71.00	2.54	0.09
ABC	210.75	8	26.34	0.94	0.49
C x Subjects within Groups	1677.38	60	27.96		

Cohort 4: EMG Baseline Levels

Cell Means

A	B	1	2	3
1	1	1.294	1.422	1.045
1	2	1.879	1.755	1.554
2	1	3.207	2.572	1.300
2	2	2.351	2.714	2.653
3	1	2.015	1.410	1.134
3	2	2.986	1.679	1.646
4	1	2.008	1.581	1.599
4	2	1.365	1.277	1.582
5	1	2.631	2.510	2.084
5	2	1.694	1.887	1.720

Summary of Analysis of Variance

Source	SS	DF	MS	F	P
Between Subjects	102.775	39			
A	15.340	4	3.835	1.43	0.249
B	0.115	1	0.115	0.04	0.838
AB	6.652	4	1.663	0.62	0.653
Subjects within Groups	80.667	30	2.689		
Within Subjects	29.005	80			
C	5.228	2	2.614	12.12	0.000
AC	4.645	8	0.581	2.69	0.013
BC	1.797	2	0.899	4.17	0.020
ABC	4.401	8	0.550	2.55	0.018
C x Subjects within Groups	12.935	60	0.216		

APPENDIX G

ANALYSIS OF DEPENDENT MEASURES ACCORDING TO AGE

Analysis of Variance on EMG Levels

Age x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	P
Between Subjects	326.25	159			
'A' Main Effects	58.44	5	11.67	7.16	0.00
Subjects within Groups	251.24	154	1.63		
Within Subjects	112.37	320			
'B' Main Effects	13.60	2	6.80	22.97	0.00
'A*B' Interaction	7.16	10	0.71	2.42	0.01
'B' x Subjects within Groups	91.16	308	0.30		

APPENDIX H

ANALYSIS OF DEPENDENT MEASURES ACCORDING TO SEX

Analysis of Variance for IPAT Scores

Sex x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	69544.00	159			
'A' Main Effects	2893.36	1	2893.36	6.86	0.01
Subjects within Groups	66650.75	158	421.84		
Within Subjects	11610.00	320			
'B' Main Effects	2070.33	2	1035.17	34.77	0.00
'A*B' Interaction	4.59	2	2.29	0.08	0.93
'B' x Subjects within Groups	9049.31	316	29.78		

Analysis of Variance for EMG Levels

Sex x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	326.25	159			
'A' Main Effects	27.24	1	27.24	14.40	0.00
Subjects within Groups	299.01	158	1.89		
Within Subjects	112.35	320			
'B' Main Effects	14.64	2	7.32	23.90	0.00
'A*B' Interaction	0.21	2	0.10	0.34	0.71
'B' x Subjects within Groups	96.73	316	0.31		

Analysis of Covariance for Sex: IPAT Scores

Test for Homogeneity of Regression

Source	DF	SS	MS	F	p
Dif	1	6.504	6.50	.072	0.78
Err	156	14032.25	89.95		

Analysis of Covariance

Source	DF	MS	Adj. F	p
Group	1	49.15	0.55	0.46
Wth	157	89.42		

R SQ = 0.5256

Group	Unadj. Mean	Adj. Mean
Male	31.88	34.57
Female	37.24	35.75

D

Analysis of Covariance for Sex: EMG Levels

Test for Homogeneity of Regression

Source	DF	SS	MS	F	p
Dif	1	.4329	0.4329	1.045	0.308
Err	156	64.63	0.4143		

Analysis of Covariance

Source	DF	MS	Adj. F	p
Group	1	3.040	7.336	0.008
Wth	157	.4144		

R SQ = 0.3516

Group	Unadj. Mean	Adj. Mean
Male	1.284	1.420
Female	1.790	1.714

APPENDIX I

ANALYSIS OF DEPENDENT MEASURES ACCORDING TO SELF-ADDRESSED ANXIETY LEVEL

Analysis of Variance on STAI-T Scores

(H,M,L x Time)

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	44095.44	159			
'A' Main Effects	3588.52	2	1794.26	7.55	0.00
Subjects within Groups	37306.88	157	237.62		
Within Subjects	9476.00	320			
'B' Main Effects	753.25	2	376.63	18.66	0.00
'A*B' Interaction	132.80	4	33.20	1.65	0.16
'B' x Subjects within Groups	6336.94	314	20.18		

Analysis of Variance on IPAT Scores

(H,M,L x Time)

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	69544.00	159			
'A' Main Effects	4197.78	2	2098.89	5.21	0.01
Subjects within Groups	63307.38	157	403.23		
Within Subjects	11610.00	320			
'B' Main Effects	757.27	2	378.63	12.64	0.00
'A*B' Interaction	6.49	4	1.62	0.05	0.99
'B' x Subjects within Groups	9408.56	314	29.96		

Analysis of Covariance for STAI-T Scores

(H,M,L x Time)

Test for Homogeneity of Regression

Source	DF	SS	MS	F	p
Dif	2	267.941	133.97	2.500	0.085
Err	154	8254.926	53.603		

Analysis of Covariance

Source	DF	MS	Adj. F	p
Grp	2	30.820	.5641	0.570
With	156	54.634		

R SQ = 0.4652

Group	Unadj. Mean	Adj. Mean
High	45.924	42.585
Medium	39.616	41.615
Low	37.875	43.940

Analysis of Covariance for IPAT Scores

(H,M,L x Time)

Test for Homogeneity of Regression

Source	DF	SS	MS	F	p
Dif	2	386.203	193.102	2.1742	0.117
Err	154	13677.491	88.814		

Analysis of Covariance

Source	DF	MS	Adj. F	p
Grp	2	12.156	0.1348	0.874
Wth	156	90.151		

R SQ = 0.5099

Group	Unadj. Mean	Adj. Mean
High	39.303	35.815
Medium	32.965	35.010
Low	28.000	34.793

APPENDIX J

ANALYSIS OF VARIANCE FOR TRAINING DATA

Analysis of Variance on Beginning EMG Levels

Group x Sessions

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	197.24	63			
'A' Main Effects	0.74	1	0.74	0.23	0.632
Subjects within Groups	196.51	62	3.17		
Within Subjects	75.96	320			
'B' Main Effects	9.04	5	1.81	8.59	0.000
'A*B' Interaction	1.65	5	0.33	1.57	0.170
'B' x Subjects within Groups	65.27	310	0.21		

Analysis of Variance on End EMG Scores

Group x Sessions

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	116.03	63			
'A' Main Effects	0.08	1	0.08	0.04	0.84
Subjects within Groups	115.95	62	1.87		
Within Subjects	77.66	320			
'B' Main Effects	5.58	5	1.12	4.98	0.00
'A*B' Interaction	2.51	5	0.50	2.24	0.05
'B' x Subjects within Groups	69.57	310	0.22		

APPENDIX K

ANALYSIS OF VARIANCE FOR AGE CATEGORIES ACROSS TRAINING SESSIONS

Analysis of Variance on Beginning EMG Levels

Age x Sessions

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	197.24	63			
'A' Main Effects	43.77	4	10.94	4.11	0.01
Subjects within Groups	157.17	59	2.66		
Within Subjects	75.96	320			
'B' Main Effects	9.53	5	1.91	9.55	0.00
'A*B' Interaction	9.30	20	0.47	2.33	0.00
'B' x Subjects within Groups	58.86	295	0.20		

Analysis of Variance on End EMG Levels

Age x Sessions

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	116.02	63			
'A' Main Effects	22.92	4	5.73	3.594	0.01
Subjects within Groups	94.06	59	1.59		
Within Subjects	77.66	320			
'B' Main Effects	5.26	5	1.05	4.685	0.00
'A*B' Interaction	7.11	20	0.36	1.585	0.05
'B' x Subjects within Groups	66.21	295	0.22		

APPENDIX L

ANALYSIS OF VARIANCE FOR MALES AND FEMALES ACROSS TRAINING SESSIONS

Analysis of Variance on Beginning EMG Levels

Sex x Sessions

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	197.24	63			
'A' Main Effects	40.75	1	40.75	16.14	0.00
Subjects within Groups	156.50	62	2.52		
Within Subjects	75.96	320			
'B' Main Effects	7.91	5	1.58	7.45	0.00
'A*B' Interaction	1.13	5	0.23	1.06	0.38
'B' x Subjects within Groups	65.79	310	0.21		

Analysis of Variance on End EMG Levels

Sex x Sessions

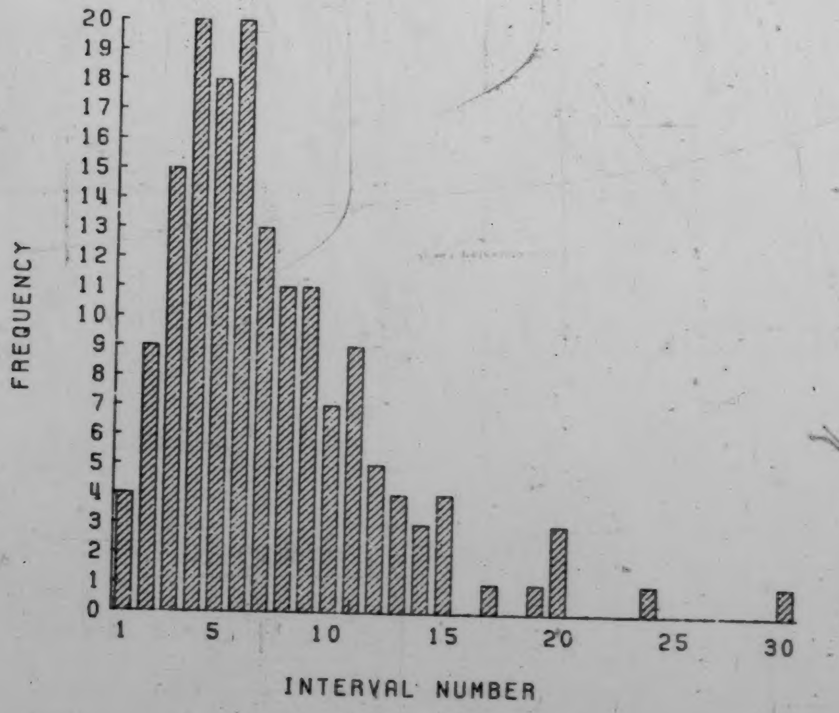
Summary of Analysis of Variance

Source	SS	DF	MS	F	P
Between Subjects	116.03	63			
'A' Main Effects	24.48	1	24.48	16.58	0.00
Subjects within Groups	91.55	62	1.48		
Within Subjects	77.66	320			
'B' Main Effects	5.37	5	1.07	4.64	0.00
'A*B' Interaction	0.32	5	0.06	0.27	0.93
'B' x Subjects within Groups	71.77	310	0.23		

APPENDIX M

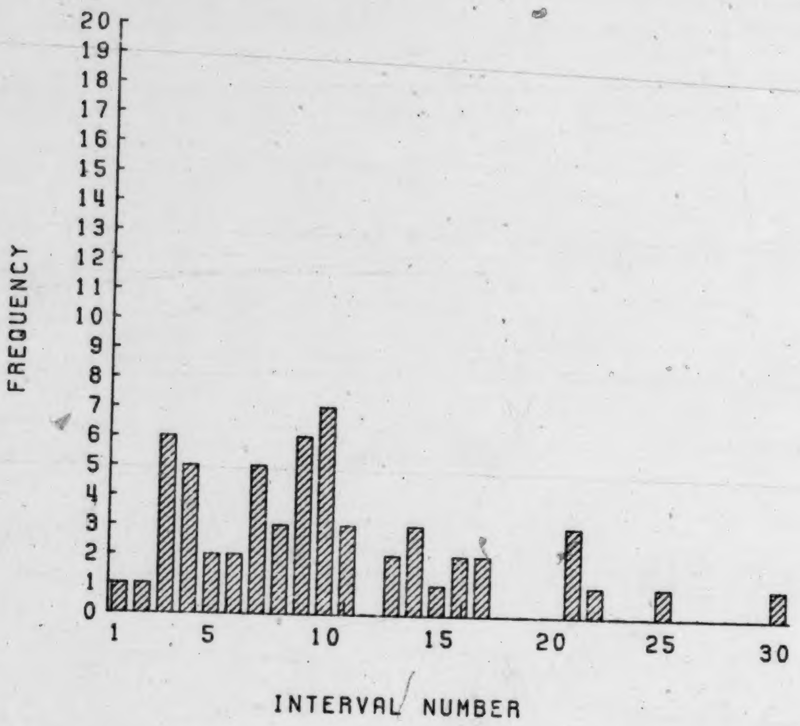
HISTOGRAMS OF RAW EMG DATA

FIVE MINUTE EMG BASELINE FOR 160 SUBJECTS



Interval		
Number	Width	
1	0.56	0.78
2	0.78	1.00
3	1.00	1.22
4	1.22	1.43
5	1.43	1.65
6	1.65	1.87
7	1.87	2.09
8	2.09	2.31
9	2.31	2.55
10	2.53	2.74
11	2.74	2.96
12	2.96	3.18
13	3.18	3.40
14	3.40	3.62
15	3.62	3.83
16	3.83	4.05
17	4.05	4.27
18	4.27	4.49
19	4.49	4.71
20	4.71	4.93
21	4.93	5.14
22	5.14	5.36
23	5.36	5.58
24	5.58	5.80
25	5.80	6.02
26	6.02	6.24
27	6.24	6.45
28	6.45	6.67
29	6.67	6.89
30	6.89	7.11

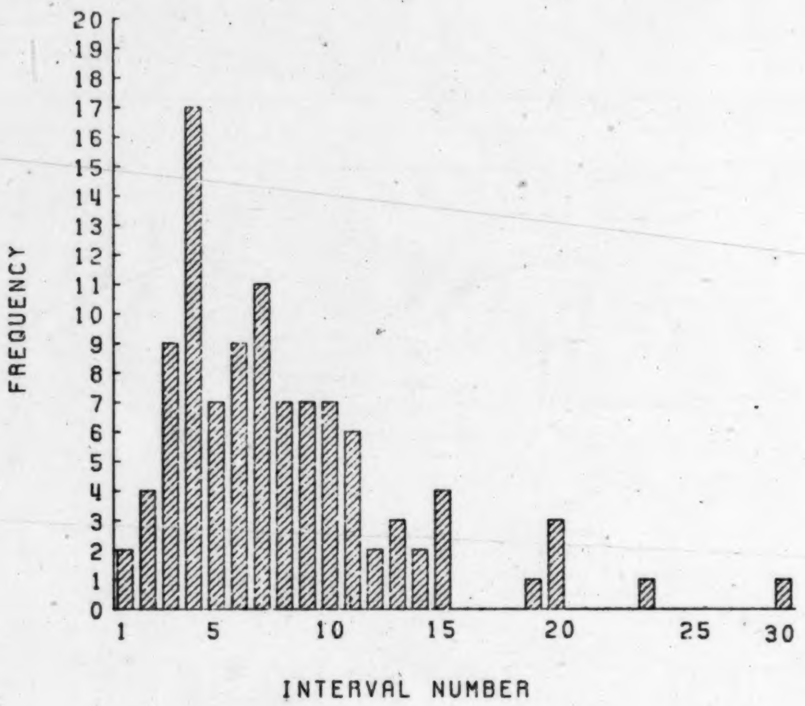
FIVE MINUTE EMG BASELINE FOR 57 MALES



Interval		
Number	Width	
1	0.61	0.73
2	0.73	0.85
3	0.85	0.97
4	0.97	1.10
5	1.10	1.22
6	1.22	1.34
7	1.34	1.46
8	1.46	1.58
9	1.58	1.70
10	1.70	1.83
11	1.83	1.95
12	1.95	2.07
13	2.07	2.19
14	2.19	2.31
15	2.31	2.44

Interval		
Number	Width	
16	2.44	2.56
17	2.56	2.68
18	2.68	2.80
19	2.80	2.92
20	2.92	3.04
21	3.04	3.17
22	3.17	3.29
23	3.29	3.41
24	3.41	3.53
25	3.53	3.65
26	3.65	3.77
27	3.77	3.90
28	3.90	4.02
29	4.02	4.14
30	4.14	4.26

FIVE MINUTE EMG BASELINE FOR 103 FEMALES



Interval		
Number	Width	
1	0.60	0.82
2	0.82	1.03
3	1.03	1.25
4	1.25	1.47
5	1.47	1.68
6	1.68	1.90
7	1.90	2.12
8	2.12	2.33
9	2.33	2.55
10	2.55	2.77
11	2.77	2.99
12	2.99	3.20
13	3.20	3.42
14	3.42	3.64
15	3.64	3.85
16	3.85	4.07
17	4.07	4.29
18	4.29	4.50
19	4.50	4.72
20	4.72	4.94
21	4.94	5.16
22	5.16	5.37
23	5.37	5.59
24	5.59	5.81
25	5.81	6.02
26	6.02	6.24
27	6.24	6.46
28	6.46	6.67
29	6.67	6.89
30	6.89	7.11

APPENDIX N

ANALYSIS OF VARIANCE FOR AWARENESS STAGE OF TREATMENT

Analysis of Variance of IPAT Scores

Group x Time

Summary of Analysis of Variance

Source	SS	DF	MS	F	p
Between Subjects	44463.75	159			
'A' Main Effects	1943.38	4	485.84	1.77	0.14
Subjects within Groups	42520.38	155	274.38		
Within Subjects	4090.50	160			
'B' Main Effects	243.25	1	243.25	10.56	0.00
'A*B' Interaction	275.75	4	68.94	2.99	0.02
'B' x Subjects within Groups	3571.50	155			